Health Canada Santé Canada
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NATIONAL AMBIENT AIR QUALITY OBJECTIVES FOR PARTICULATE MATTER

PART 1: SCIENCE ASSESSMENT DOCUMENT

A report by the
CEPA/FPAC Working Group
on Air Quality Objectives
and Guidelines

Canadä

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This report is published under Section 8 of the Canadian Environmental Protection Act (CEPA).

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PREFACE

The Canadian Environmental Protection Act (*CEPA*), passed into law in 1988, replaces and builds upon the Clean Air Act and the Environmental Contaminants Act. The opening statement of the Act declares that "the protection of the environment is essential to the well-being of Canada". *CEPA* allows the federal government to assess substances and control their impact through national environmental quality objectives, guidelines, codes of practice, and/or regulations.

Provincial governments have the primary responsibility in many areas of air pollution control, with federal actions integrated with those of the provinces. The *CEPA* Federal/Provincial Working Group on Air Quality Objectives and Guidelines, consisting of representatives of federal, provincial and territorial departments of environment and health, reviews and recommends ambient air quality objectives.

Canada's *National Ambient Air Quality Objectives(s)* prescribe targets for air quality, measured at the relevant receptor (persons, plants, animals, materials). *National Ambient Air Quality Objectives are national goals[1] for outdoor air quality that protect public health, the environment, or aesthetic properties of the environment.* The development of NAAQOs involves first a scientific review of the physical and chemical properties of a substance, its sources, environmental, animal and human health effects, and environmental and human exposure assessment, and secondly, integration of this information within a framework of risk assessment. The Science Assessment Document contains this critical scientific evaluation, and lays the scientific groundwork for establishing the air quality objectives. Reference Levels, levels above which there are demonstrated effects on human health and/or the environment, are identified. A document outlining the process followed in reviewing and interpreting the scientific information is published separately[1]. This document contains the scientific evaluation of Particulate Matter.

National Ambient Air Quality Objectives are for focussing air quality management strategies and plans. The derivation of these targets may consider some element of benefit/risk analysis, reflecting a philosophy of environmental health protection and long term risk reduction while recognizing technological and economic limits. The broad range of potential responses by the population, the ecosystems, and organisms in the environment are considered. Given the range of these sensitivities however, the resulting objectives may not protect all.

The process[2] of establishing National Ambient Air Quality Objectives is a dynamic and continuous one. The air quality objectives are established to provide the current state of knowledge about an air quality parameter, a uniform scale for assessing the quality of air in all parts of Canada, and guidance to governments for making risk management decisions such as planning control strategies and setting local standards.

It is recognized that not all locations in Canada will meet these air quality objectives immediately, or at all times, and that priority given to meet these values may be based on factors such as available control technology, costs, benefits, and the degree to which the recommended objectives are exceeded. The expectation is that strategies will be implemented to facilitate the reduction of ambient air concentrations to a level at or below the air quality objective(s) as soon as practicable. The principles of continuous improvement and nondegradation of environmental quality are advocated.

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[1] A Protocol for the Development of National Ambient Air Quality Objectives Part 1: Science Assessment Document and Derivation of the Reference Levels, WGAQOG, 1996.

[2] A draft document, Protocol for the Development of National Ambient Air Quality Objectives. Part 2, outlines the process for developing air quality objectives.

NOTE:

In January 1998, Canadian Environment Ministers (with the exception of Quebec) signed the Canada-Wide Accord on Environmental Harmonization and its sub-agreement on Canada-Wide Standards (CWSs). The CWS process provides new tools for the management of environmental issues of national interest. Recognizing that both NAAQOs and CWSs have a role to play in the management of air quality, federal, provincial, and territorial health and environment departments have integrated the NAAQO and CWS processes. Air pollutants that have been identified by governments as needing to be managed will be targeted for either CWS or NAAQO development, not both.

In January 1998, Environment Ministers identified particulate matter as a priority for Canada-Wide Standards. As a result, federal, provincial, and territorial health and environment departments agreed that NAAQOs for particulate matter will no longer be developed. Rather, this Science Assessment Document will form the Risk Assessment report for the development of CWSs for particulate matter.

Availability of this Particulate Matter Science Assessment Document has been announced in the *Canada Gazette* Part I. This report is published under Section 8 of the *Canadian Environmental Protection Act* (CEPA).

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ABBREVIATIONS

ACRONYMS

AHH	Aryl hydrocarbon hydroxylase
AISI	American Iron and Steel Institute
BAL	Bronchoalveolar lavage
BALT	Bronchus-associated lymphatic tissue
BaP	Benzo[a]pyrene
BS	British Smoke Shade or Black Smoke
CAAMP	Canadian Acid Aerosol Measurement Program
CARE	Centre for Atmospheric Research Experiments
CCN	Cloud condensation nuclei
CEPA	Canadian Environmental Protection Act
CHIEF	Clearing House for Inventory and Emission Factors
CI	Confidence interval
CMB	Chemical mass balance
CMD	Count median diameter
CoH	Coefficient of Haze
COPD	Chronic obstructive pulmonary disease
DRUM	Davis Rotating-drum Universal-size-cut Monitoring
EC	Elemental carbon
EPA	Environmental Protection Agency
EPS	Environmental Protection Service
ERDA	Energy Research Development Authority
ESP	Electrostatic precipitator
ETS	Environmental tobacco smoke
FAC	Fractional aerosol coefficient
FAM	Fixed ambient monitor
FEF$_{25-75}$	Mean forced expiratory flow during the middle half of forced vital capacity
FEV$_1$	Forced expiratory volume in one second
FP	Fine particles
FPAC	Federal–Provincial Advisory Committee
FRG	(former) Federal Republic of Germany
FVC	Forced vital capacity
GVRD	Greater Vancouver Regional District
HIS	Health Interview Survey
IADN	International Acid Deposition Network
IASI	Indoor Air Sampling Impactor
ICD	International Classification of Disease
Ig	Immunoglobulin
IL	Interleukin
IMPROVE	Interagency Monitoring of Protected Visual Environments
IP	Inhalable particulates
ISWS	Illinois State Water Survey
LD$_{50}$	Lethal dose for 50% of the test population
LFV	Lower Fraser Valley
LOEL	Lowest-observed-effect level
MAMU	Mobile Air Monitoring Unit
MDL	Minimum detectable limit
MEF$_{25\%}$	Maximum flow at 25% of vital capacity

MMAD	Mass median aerodynamic diameter
MMD	Mass median diameter
MMEF	Maximum mid-expiratory flow
MMT	Methylcyclopentadienyl manganese tricarbonyl
MOUDI	Micro Orifice Uniform Deposit Impactor
MTD	Maximum tolerated dose
NAAQO	National Ambient Air Quality Objective(s)
NAAQS	National Ambient Air Quality Standard
NAPS	National Air Pollution Surveillance
NRCC	National Research Council of Canada
NSSC	Neutral sulphite semi-chemical
NTP	National Toxicology Program
OC	Organic carbon
OECD	Organisation for Economic Co-operation and Development
PAH	Polycyclic aromatic hydrocarbon
PAN	Peroxyacetyl nitrate
PCA	Principal component analysis
PEC	Particulate elemental carbon
PEF	Peak expiratory flow
PEM	Personal exposure monitor
PM	Particulate matter
$PM_{2.5}$	Particulate matter 2.5 µm or less in diameter
PM_{10}	Particulate matter 10 µm or less in diameter
PMN	Polymorphonuclear leukocytes
POM	Polycyclic organic material
PREVENT	Pacific Northwest Regional Visibility Experiment Using Natural Tracers
P-TEAM	Particle Total Exposure Assessment Methodology
REVEAL	Regional Visibility Experimental Assessment in the Lower Fraser Valley
RR	Relative risk
RSP	Respirable suspended particulate
RWC	Residential wood combustion
SAM	Stationary ambient monitor
SCAQS	Southern California Air Quality Study
SIM	Stationary indoor monitor
SIP	State Implementation Plan
SMSA	Standard Metropolitan Statistical Area
SSI	Size selective inlet
TEOM	Tapered Element Oscillation Microbalance
THEES	Total Human Environmental Exposure Study
TNF	Tumour necrosis factor
TP	Thoracic particles
TSP	Total suspended particulate
UQM	Upper quartile mean
VAQS	Valley Air Quality Study
VKmT	Vehicle–kilometres travelled
VOC	Volatile organic compound
V/Q	Ventilation–perfusion ratio
VRP	Visibility Reducing Particles
WHO	World Health Organization

CHEMICAL COMPOUNDS

CO	Carbon monoxide
Fe_3O_4	Black iron oxide
Ga_2O_3	Gallium oxide
H^+	Hydrogen ion
H_2SO_4	Sulphuric acid
HCl	Hydrochloric acid
HNO_3	Nitric acid
K_2CO_3	Potassium carbonate
N_2O_5	Dinitrogen pentoxide
NaCl	Sodium chloride
NH_3	Ammonia
NH_4Cl	Ammonium chloride
NH_4HSO_4	Ammonium bisulphate
NH_4NO_3	Ammonium nitrate
$(NH_4)_2SO_4$	Ammonium sulphate
NO	Nitric oxide
NO_2	Nitrogen dioxide
NO_3^-	Nitrate
NO_x	Nitrogen oxides
O_3	Ozone
OH•	Hydroxyl radical
SO_2	Sulphur dioxide
SO_4^{2-}	Sulphate
SO_x	Sulphur oxides

UNITS

cfm	cubic feet per minute
cm	centimetre
cm^2	square centimetre
cm^3	cubic centimetre
d	day
g	gram
Gg	gigagram
h	hour
km	kilometre
kt	kilotonne
L	litre
µg	microgram
µm	micrometre
m^2	square metre
m^3	cubic metre
mg	milligram
min	minute
mL	millilitre
mm	millimetre
Mt	megatonne
nm	nanometre
s	second
wk	week
yr	year

1 INTRODUCTION

The *Canadian Environmental Protection Act (CEPA)* Federal/Provincial Working Group on Air Quality Objectives and Guidelines (WG) is directed by the *CEPA* Federal-Provincial Advisory Committee (FPAC) to develop National Ambient Air Quality Objectives (NAAQOs) for airborne pollutants and to undertake their periodic reassessment. NAAQOs are established to provide a measure of protection to public health and the environment from adverse effects due to airborne pollutants.

Air Quality Objective (AQO) development begins with a scientific review that is embodied within this document, the Science Assessment Document. The evaluation of available scientific data is followed by the identification of Reference Levels through appropriate analyses as outlined in "A Protocol for the Development of National Ambient Air Quality Objectives: Part One: Science Assessment Document and Derivation of the Reference Level(s)" (CEPA/FPAC WGAQOG, 1997).The Reference Level is a level above which there are demonstrated effects on a receptor (human or environmental). More than one form of Reference Level can be identified (e.g., 24 h, SUM60) for one or more receptors. The availability of control technology, the potential economic impacts associated with reducing ambient air pollutant concentrations, public/stakeholder consultation on AQO options, and other considerations, are introduced during the derivation of the Air Quality Objective. Several options are considered, including concentrations higher, lower or equivalent to the Reference Level, depending on ambient background concentrations, uncertainties in the underlying scientific data, and the additional factors listed above.

Canadian air quality objectives for total suspended particulate matter (TSP) were first developed in the 1970s and were revised in the 1980s. At that time, three ranges of air quality objectives were formulated – "desirable", "acceptable" and "tolerable". The TSP objectives are based upon earlier scientific research, before the late 1960s, when many air pollution episodes resulted in severe health effects – leading to several thousand premature deaths over a period of a few days, in cities such as London, England. Governments of the day took steps to reduce pollution levels, consisting primarily of combustion-related particulate matter (PM) usually combined with sulphur dioxide, and episodes of severe air pollution similar to the London fog episodes have now become rare.

The TSP air quality objectives, like those developed in the US, Britain and Europe, were based upon the assumption that low ambient concentrations of PM measured in North America and Western European cities were not associated with any adverse effects on health or the environment. Beginning about 1990, an avalanche of scientific studies carried out in the US, Canada, Britain and Europe cast serious doubt on this assumption. The studies demonstrated adverse effects on cardiorespiratory health, including both increased hospitalizations and increased premature mortality, associated with ambient PM levels that were often far below the current TSP objectives. These findings prompted reexamination of the objectives, not only in Canada, but also in the US, Britain, the European Community, and the World Health Organization.

This Science Assessment Document represents the Canadian reexamination of particulate matter air pollution and its relationship to increases in health and environmental effects. Rather than discard the TSP objectives, which the Working Group acknowledges are outdated both temporally and scientifically, new air quality objectives for particulate matter equal to or less than 10 microns in diameter (PM_{10}) and its fine subfraction ($PM_{2.5}$) are being developed. The evaluation focusses on the smaller particles, as compared to total suspended particles that range in size from 0.005 μm up to 100 μm, because the recent evidence shows that particle size is important. Size influences the site of deposition in the human respiratory tract and the consequent degree of toxicity that may be experienced. Particle size also reflects the origin and formation of airborne particles; larger sized particles are often of crustal origin and smaller particles come from combustion processes.

In addition, it is the fine fraction that contributes to reduced visibility in the atmosphere.

Included in this document is a description of PM: physical and chemical characteristics, monitoring methods, sources and behaviour in the environment, background and ambient concentrations, exposure of Canadians, and an evaluation of the available toxicological information and data. This is followed by the risk characterization for human and environmental receptors and the development of Reference Levels based on the scientific information.

2 PHYSICAL AND CHEMICAL CHARACTERISTICS OF PM

"Particulate matter" or "PM" refers to all airborne solid and liquid particles – except pure water – that are microscopic in size. Particle diameters may range from approximately 0.005 µm to 100 µm, although the suspended portion generally consists of particles less than 40 µm. Particulate matter is unique among atmospheric constituents in that it is not defined based on its chemical composition. It may include a broad range of chemical species, such as elemental and organic carbon compounds; oxides of silicon, aluminum and iron; trace metals; sulphates; nitrates and ammonium.

Several terms have been used to describe types of particulate matter. Table 2.1 defines some of the more common terms. The fraction of particulate matter that is currently included under federal air quality objectives in Canada is total suspended particulate matter (Canada Gazette, 1989), which by general agreement, refers to particles with diameters less than 100 µm and which ". . . excludes substances with a known specific toxic effect (arsenic, asbestos, benzo-a-pyrene, beryllium, sulphuric acid mist, etc.) and refers only to those which are chemically relatively inert" (SAQO, 1976).

The current discussion will focus on PM_{10} and its fine subfraction, $PM_{2.5}$. The term "PM_{10}" refers to particles that are 10 µm or less in diameter. Particles of this size range are also called "thoracic particles," reflecting the fact that they can be inhaled into the tracheobronchial and alveolar (thoracic) regions of the respiratory system. They are also sometimes referred to as "inhalable particles", although this term is generally applied to particles smaller than 15 µm in diameter. PM_{10} is generally subdivided into a fine fraction ($\leq$2.5 µm) or less in diameter ($PM_{2.5}$) and a coarse fraction (>2.5 µm) in diameter, reflecting its typically *bimodal* distribution. Sometimes, the fine fraction refers to particles less than 2.0 µm in diameter; for this report, "fine" will refer to 2.5 µm or less. These particles are sometimes referred to as "respirable particles".

Particles are further classified as "primary" or "secondary" in nature. Primary particles are those emitted directly into the atmosphere, whereas secondary particles are formed through physical or chemical transformations that take place in the atmosphere. Primary PM_{10} may result from natural or human (anthropogenic) activities. Natural sources include windblown dust, sea spray and wildfires. Anthropogenic sources include fuel combustion, industrial processes and transportation. High-temperature combustion sources contribute to the fine fraction of PM_{10}, whereas particles produced by grinding activities or wind erosion are predominantly found in the coarse fraction. Secondary particles are predominantly found in the fine fraction, and can also be of either natural or anthropogenic origin. They typically result from reactions involving such precursor gases as sulphur dioxide (SO_2), nitrogen oxides (NO_x), ammonia (NH_3) and various organic gases, yielding particle sulphate, nitrate and condensed organic compounds.

The behaviour of particulate matter in the atmosphere and its potential to affect human health and atmospheric visibility are dependent on its' physical and chemical characteristics. These characteristics, in turn, are dependent on the source type and on formation processes that particles undergo at the source or in the atmosphere. In the following sections, the major physical and chemical characteristics of PM will be described. Further discussion of the characteristics associated with particulate emissions from specific sources can be found in Chapter 4. For a more detailed treatment of particulate matter, the reader is referred to the US Environmental Protection Agency (EPA) criteria document for particulate matter (US EPA, 1982a; 1996). For comparison purposes, the reader is also referred to the National Research Council of Canada (NRCC) document (NRCC, 1982) for a Canadian perspective.

2.1 PARTICLE SIZE

Particle size is considered the most important parameter in characterizing the physical behaviour of particulate matter, as it affects removal processes, atmospheric residence times and the contribution of light scattering to visibility degradation. Particle size is typically defined in terms of its diameter. Although

Table 2.1 Terminology Relating to Atmospheric Particles (after Seinfeld, 1986)

Terminology	Definition[1]
Aerosols, aerocolloids, aerodisperse systems	Tiny particles dispersed in gases.
Dusts	Suspensions of solid particles produced by mechanical disintegration of material such as crushing, grinding, and blasting. $D_p > 1.0$ µm.
Fog	A loose term applied to visible aerosols in which the dispersed phase is liquid. Usually, a dispersion of water or ice.
Fume	The solid particles generated by condensation from the vapour state, generally after volatilization from melted substances, and often accompanied by a chemical reaction such as *oxidation*. Often the material involved is noxious. $D_p < 1$ µm.
Hazes	An aerosol that impedes vision and may consist of a combination of water droplets, pollutants, and dust. $D_p < 1$ µm.
Mists	Liquid, usually water as particles suspended in the atmosphere at or near the surface of the earth; small water droplets floating or falling, approaching the form of rain, and sometimes distinguished from fog as being more transparent or as having particles perceptibly moving downward. $D_p > 1$ µm.
Particle	An aerosol particle may consist of a single continuous unit of solid or liquid containing many molecules held together by intermolecular forces and primarily larger than molecular dimensions (>0.001 µm). A particle may also be considered to consist of two or more such unit structures held together by interparticle adhesive forces such that it behaves as a single unit in suspension or upon deposit.
Smog	A term derived from smoke and fog, applied to extensive contamination by aerosols. Now sometimes used loosely for any contamination of the air.
Smoke	Small gas-borne particles resulting from incomplete combustion, consisting predominantly of carbon and other combustible material, and present in sufficient quantity to be observable independently of the presence of other solids. $D_p \geq 0.01$ µm.
Soot	Agglomerations of particles of carbon impregnated with "tar," formed in the incomplete combustion of carbonaceous material.

[1] In the list of definitions, D_p refers to the equivalent diameter of a spherical particle of unit density that has the same settling velocity as the collected particle.

liquid aerosol particles are nearly always spherical, and solid particles are often irregularly shaped, equivalent diameters are used to correct for these deviations from sphericity.

The *aerodynamic diameter* is the most commonly used expression for particle size. Based on Stokes' law, which describes the forces acting on a falling sphere in a stagnant medium, it defines the equivalent diameter of a spherical particle that has the same settling velocity as the collected particles. If the density of the collected particles is known, the equivalent diameter D_p is referred to as the Stokes' diameter. If it is not known, a particle density of 1 g/cm^3 is assumed, and the equivalent diameter is referred to as the equivalent aerodynamic diameter. Stokes' Law is applicable to particles with diameters between 1 and 100 µm. Below 1 µm, the assumption that the airstream in contact with the particle has the same velocity as the particle is no longer valid, and the Cunningham correction factor must be applied to account for particle slip between air molecules. This factor is estimated to vary with the nature of the particle surface, with reported values of 0.7 for rough spheres, 0.82-0.9 for aqueous solution drops and 0.87 for oil drops (Fuchs, 1964).

Because particle size may vary over four orders of magnitude, characterizing particulate matter in terms of particle distributions is convenient and, more specifically, a continuum of size variations. The total number of particles is defined as

$$N = \int_0^\infty n(D_p)dD_p \qquad (2.1)$$

where $n(D_p)$ represents the size distribution function for particle size. A normalized size distribution function is then defined as $\tilde{n}(D_p)=n(D_p)/N$, where $\tilde{n}(D_p)dD_p$ is the fraction of the total number of particles per cubic metre of air having diameters in the range of D_p to D_p+dD_p.

Until the early 1970s, the two most common types of frequency distributions used by atmospheric scientists were Junge's (1955) power law distribution for particle size and the log-normal distribution for mass. Junge's power law is of the form

$$\frac{dN}{dD} = AD^{-k} \tag{2.2}$$

where A and k are constants. Although it was valid for number distributions, Clark and Whitby (1967) found this function to be inadequate for describing surface and volume distributions. Limitations were also identified in the log-normal distribution (Fuchs, 1964), in that it was adequate only when particulate matter was sampled perfectly.

Recognizing the weaknesses with the two established methods, Whitby et al. (1972) instead expressed the size distributions in terms of the logarithm of the diameter, obtaining

$$\frac{dN}{d\log D_p} = n(\log D_p) \tag{2.3}$$

for number distributions,

$$\frac{dS}{d\log D_p} = n_s(\log D_p) \tag{2.4}$$

for surface area distributions and

$$\frac{dV}{d\log D_p} = n_v(\log D_p) \tag{2.5}$$

for volume (or mass) distributions, where S and $n_s(\log D_p)$ are surface area and surface area distribution function, and V and $n_v(\log D_p)$ are volume and volume distribution function, respectively. An advantage of presenting the data in this form is that the area under the curve is proportional to the quantity (number, surface area or volume) in the given size interval. For a more detailed description of particle size distributions, the reader is referred to Seinfeld (1986).

A well-known example of number, surface area and volume distributions obtained by Whitby (1975) during a smog episode in Pasadena, California, in 1969 is presented in Figure 2.1. The respective ordinates are $dN/d\log D_p$, $dS/d\log D_p$ and $dV/d\log D_p$, and the abscissa is $\log D_p$. Figure 2.1 shows that in an urban environment, size distributions may be multimodal, depending on the quantity involved. Number distributions exhibit a single mode, with a maximum near 0.01 µm. Surface area distributions exhibit one or two modes, with a primary maximum near 0.2 µm. Volume or mass distributions are characterized by two modes, with maxima near 0.3 and 10 µm, and a third mode if sampling takes place near a strong source of fresh nuclei.

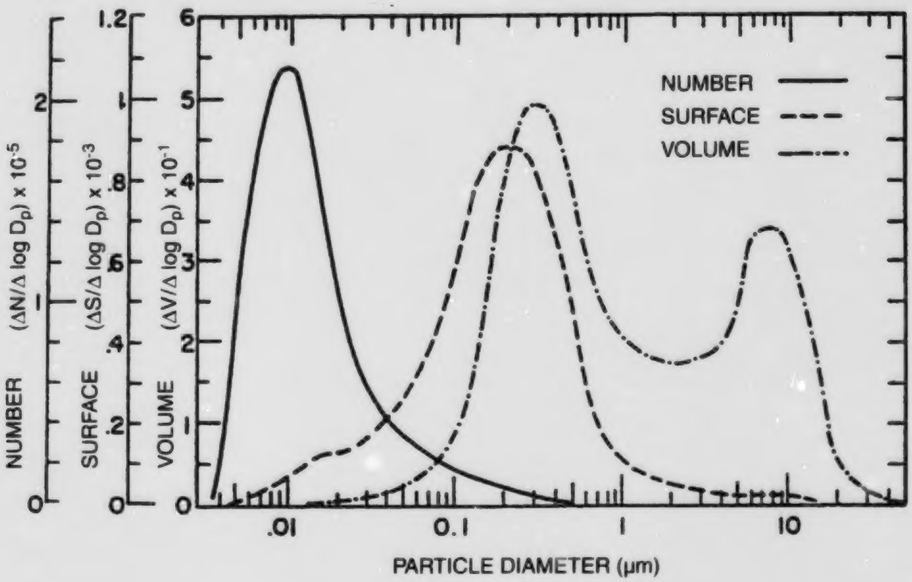
These modes reflect the different source types and the different transformation mechanisms at work in the atmosphere. They are generally identified as follows:

(a) nuclei mode: $D_p < 0.1$ µm,
(b) accumulation mode: 0.1 µm $< D_p <$ 2 µm, and
(c) *sedimentation* or coarse mode: $D_p > 2$ µm.

Particles in the nuclei mode ($D_p < 0.1$µm), also called Aitken nuclei, are formed primarily from the condensation of hot vapours during combustion processes and from the *nucleation* of atmospheric species to form new particles. Although the greatest concentration of airborne particles is found in the nuclei mode, these particles contribute little to overall particle mass loading. They are subject to random or *Brownian motion* and to *coagulation* processes in which particles collide to yield larger particles. Therefore, the atmospheric residence time of nuclei mode particles is very short.

It has been estimated that the concentration of particles of 0.01 µm average diameter, initially present at levels typical of urban air (10^5 particles per cubic centimetre; or $10^5/\text{cm}^3$), should decrease by a factor of two within about 30 minutes owing to coagulation (Wallace & Hobbs, 1977). This compares with an estimated time of 500 hours for a particle of 0.2 µm diameter initially present in concentrations of $10^3/\text{cm}^3$. Because of rapidly decreasing particle mobility as particle size increases, coagulation processes are limited to particles with diameters less than 0.2 µm.

Figure 2.1 Normalized Frequency Plots of Number, Surface and Volume Distributions for Pasadena Smog in 1969 (from Whitby, 1975)



Particles found in the accumulation mode (0.1 µm < D_p < 2 µm) result from the coagulation of particles in the nuclei mode and from the condensation of vapours onto existing particles that then grow into this size range. The accumulation mode accounts for most of the particle surface area and a sizable portion of the particle mass. It is so-named because removal processes are least efficient in this size range. The atmospheric lifetime of particles in the accumulation mode may be on the order of days to weeks.

The coarse particle mode (D_p > 2 µm) is typically associated with mechanical processes, such as wind erosion that produces dust particles, industrial processes that produce fly ash, and breaking ocean waves that produce sea spray. These processes generally lack the energy to produce very fine particles, as one would find in high-temperature combustion processes. Particles in the coarse mode are efficiently removed by gravitational settling and, as a result, are characterized by short atmospheric residence times, on the order of hours to days. Therefore, although the coarse mode accounts for much of the total mass of ambient PM_{10}, it contributes little to the total number of particles. As a result, little mass is transferred from the accumulation mode to the coarse mode. The coarse mode also contributes little to particle surface area distributions, owing to the small numbers and the small surface area to volume ratios of particles relative to those in the accumulation mode.

Figure 2.2 shows normalized particle number distributions obtained from measurements in several different environments. Urban settings, which are associated with heavy anthropogenic activity, are seen to contain the greatest number of fine particles, whereas desert dust storms contain a large number of giant particles. Variations in particle concentrations are greatest for the smaller and larger particles, reflecting their shorter residence times. To maintain the observed concentration levels, there must be a continuous production of very small and large particles in the atmosphere. All distributions show a rather uniform decrease in concentration beyond a particle size of approximately 0.1 µm, thus masking the fact that considerable particle mass lies at particle sizes greater than 2.5 µm.

To represent contributions to the coarse fraction better, normalized particle volume distributions based on the same data as the aforementioned number distributions are shown in Figure 2.3. Urban particulate matter is shown as exhibiting high mass concentrations in both the fine and coarse fractions. Recent studies over eastern North America confirm that away from immediate sources, particle mass distributions are dominated by particles in fine (0.2-0.5 µm) and coarse (5-30 µm) modes (Leaitch & Isaac, 1991). An exception is near large-particle sources such as the ocean.

Figure 2.2 Number/Size Distributions Obtained from a Large Set of Measurements from Various Environments (from Jaenicke, 1986)

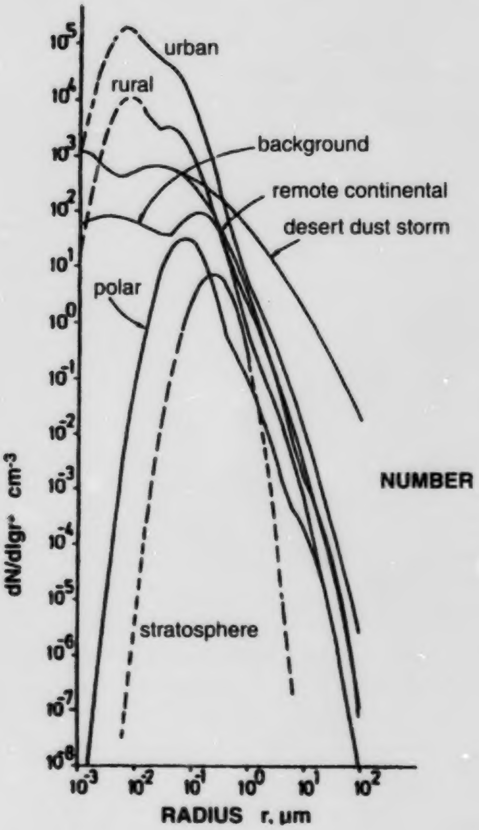
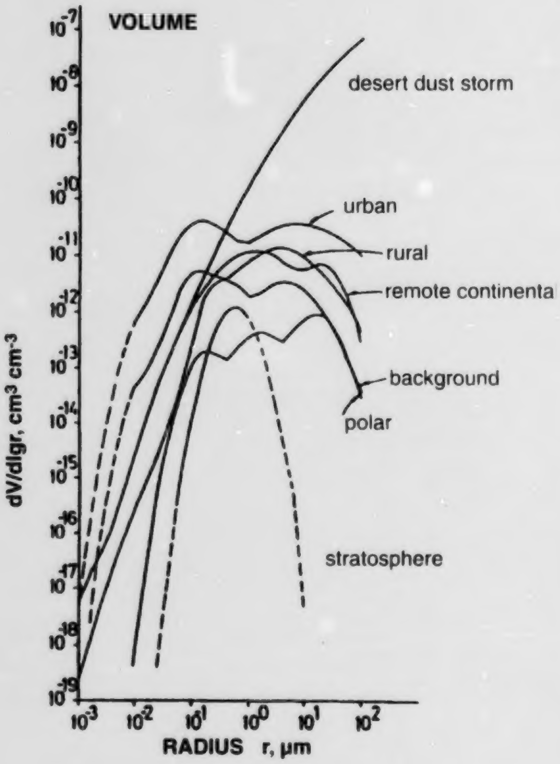


Figure 2.3 Volume Distributions Obtained from a Large Set of Measurements from Various Environments (from Jaenicke, 1986)



2.2 PARTICLE SHAPE AND DENSITY

Particle shape depends on the source type as well as the processes that a particle may undergo once in the atmosphere. Those particles formed by processes of *comminution* (grinding or pulverizing), *attrition* (abrasion or friction) or disintegration will resemble the parent material, while those particles formed by condensation or nucleation will be spherical or of similar simple shape. Condensation is often followed by solidification and formation of chain-like aggregates. Further changes to particle shape may result through processes of *crystallization*, *hydration* (addition of water molecules) and *agglomeration* (formation of a larger, permanent mass in which the individual particles can still be identified). In Table 2.2, the main categories of particle shape are identified and related to particle source type and contribution to total sample size.

Any given shape may include fine and coarse fractions from the same source type of material, (e.g., fly ash). Particles observed in urban areas are highly irregular in shape (Katrinak et al., 1993). Combustion particles also tend to be highly irregular in shape, although exposure conditions (e.g., combustion temperature) will modify the particle shape. Estimates of the specific surface area of coal fly ash are 3-20 times lower than experimental values when the particles are assumed to be nonporous spheres (Rothenberg, 1980).

Particle density (mass per unit volume) will affect both the motion and behaviour of a particle in the atmosphere. The density of airborne particles has been estimated to range from 0.5-6.5 g/cm^3 (Corn, 1976b). Those particles formed through attrition will have the same density as the parent material, whereas processes of oxidation, hydration and

Table 2.2 Particle Shapes (from Stern, 1976)

Shape	Percent by Weight in Sample		Particle Source Types
	Range	Average	
spherical	0-20	10	smoke, pollen, fly ash
irregular/ cubical	10-90	40	mineral, cinder
flakes	0-10	5	mineral, epidermis
fibrous	3-35	10	lint, plant fibre
condensation flocs	0-40	15	carbon, smoke, fume

Table 2.3 Particle Densities of Metal Oxide Agglomerates (from CARB, 1982, based on Whytlaw-Gray and Patterson, 1932)

Material	Floc Density (g/cm^{-3})	True Density (g/cm^{-3})
Silver	0.94	10.5
Mercury	1.70	13.6
Cadmium Oxide	0.51	6.5
Magnesium Oxide	0.35	3.6
Mercuric Chloride	1.27	5.4
Arsenic Trioxide	0.71	3.7
Lead Monoxide	0.62	9.4
Antimony Trioxide	0.63	5.6
Aluminum Oxide	0.18	3.7
Stannic Oxide	0.25	6.7

agglomeration will alter particle density. Those particles containing many void spaces, as is typical of agglomerates, may have a density less than one-tenth that of the parent material, as shown in Table 2.3, which compares floc or apparent particle density with the true density of the parent material.

2.3 BULK MATERIAL PROPERTIES

The physical state and growth of particles in the atmosphere are affected by such bulk material properties as chemical composition, vapour pressure, hygroscopicity and deliquescence (US EPA, 1982a). Refractive indexes, together with particle size, will affect particle scattering of light and therefore atmospheric visibility.

Because the hygroscopic or water-attracting nature of particles affects such characteristics as particle size, shape, refractive index, pH and reactivity, further discussion of this property is warranted. Strictly hygroscopic compounds such as sulphuric acid (H_2SO_4) *monotonically* increase in size with increasing relative humidity. Particles containing deliquescent *salts* such as ammonium bisulphate (NH_4HSO_4) and ammonium sulphate [$(NH_4)_2SO_4$] remain dry with increasing relative humidity until the deliquescence point, which corresponds to the water vapour pressure of the saturated salt solution, is reached. This transition is marked by a sudden uptake of water and a corresponding increase in particle size to form droplets of concentrated solution. Laboratory and field experiments show that sulphate particle size increases with increasing relative humidity (Koutrakis and Kelly, 1993). Growth rates of 50% in the mass median aerodynamic diameter (MMAD) of sulphate-, nitrate- and ammonia-containing particles (from 0.27 to 0.42 μm) have been observed as relative humidity is increased from 40 to 70% (Countess et al., 1981). As light scattering is most efficient for particles in the size range of 0.1-1.0 μm, reaching a maximum between 0.3 and 0.7 μg, particle growth through deliquescence has a large potential impact on atmospheric visibility.

Particles may also undergo a sudden release of water with decreasing relative humidity. This is termed *efflorescence*, and the associated relative humidity is called the efflorescence point. Deliquescence and efflorescence points for various compounds are presented in Table 2.4. The latter typically occur at relative humidities 30% below the deliquescence point (US EPA, 1982a).

An example of a deliquescent compound is $(NH_4)_2SO_4$, the fully neutralized ammonium salt of H_2SO_4, which undergoes a solid-liquid transition at about 80% relative humidity. Below this point, the oxidation of SO_2 to H_2SO_4 takes place predominantly in the gas phase, with H_2SO_4 condensing onto particle matter. Above this point, SO_2 readily dissolves in the sulphate droplets and is oxidized to form particle sulphate. Further discussion of this mechanism can be found in Section 2.7.

Table 2.4 Deliquescence and Efflorescence Points of Salt Particles (based on US EPA, 1982a, Countess et al., 1981)

Composition	Deliquescence (% RH)	Efflorescence (% RH)
NaCl	75.7	43
KCl	84.3	53
NaCl-KCl	73.8	38
$(NH_4)_2SO_4$	79.5	~36
$(NH_4)_3H(SO_4)_2$	69.0	–
$(NH_4)HSO_4$	39.0	–
$2NH_4NO_3{\bullet}(NH_4)_2SO_4$	56.4	–
NH_4NO_3	62.0	–

2.4 PARTICLE SURFACE PROPERTIES

Small particles are characterized by a large surface area relative to their mass, which is further increased with the presence of surface irregularities and internal pores. Under most atmospheric conditions, particles smaller than 0.1 μm diameter are known to contain significant amounts of water. Therefore, gas-liquid phase interactions are considered important on these small particles (Lodge et al., 1981).

Other surface properties of importance include electrostatic charging, adhesion and the influence of surface films (US EPA, 1982a). Electrostatic charging and adhesion can provide means of particle detection, measurement and collection. Organics such as carboxylic acids, alcohols, esters and aldehydes can form coatings on fine particles (Gill et al., 1983). As many of these coatings are surface active (surfactants), they may influence how the particle grows in a humid environment. Exposure to organic gases appears to retard the evaporation of water droplets, thereby increasing their persistence in the atmosphere (Chang et al., 1980). Carbon black particles, which are normally hydrophobic (i.e., have an aversion to water), were found to absorb large amounts of water (20-40% of dry particle mass) when coated with an organic surfactant (Andrews and Larson, 1993).

2.5 PARTICLE FORMATION AND GROWTH

Two basic processes are involved in particle formation: grinding or atomization, and nucleation of *supersaturated* vapours. Grinding and atomization are mechanical processes that result in the direct discharge of particles to the atmosphere. Owing to the nature of the formation process, these particles are typically found in the coarse mode. Examples include windblown dust and sea spray.

Nucleation of supersaturated vapours results from the molecular agglomeration of supersaturated vapours, either without foreign particles and ions (homogeneous nucleation) or in their presence (heterogeneous nucleation). Examples of homogeneous nucleation include the formation of H_2SO_4 by reaction of the hydroxyl radical (OH) with SO_2 and the formation of carboxylic acids by reaction of ozone with olefins. The condensation of hydrocarbon vapours onto carbon particles during the cooling of automobile exhaust and the condensation of H_2SO_4 molecules onto fly ash during the cooling of power plant plumes are examples of heterogeneous nucleation.

Particle growth may occur through particle-particle interactions or gas-particle interactions. Particle-particle interactions involve the coagulation of two particles to form one larger particle. Gas-particle interactions include the growth of hygroscopic particles such as sulphates and nitrates through absorption of water vapour; the absorption and adsorption of pollutant gases such as SO_2, nitrogen dioxide (NO_2) and hydrocarbons, followed by chemical reactions to yield such products as sulphate (SO_4^{2-}), nitrate (NO_3^-)and organic compounds; and the condensation of low-vapour-pressure molecules formed in gas-phase reactions such as H_2SO_4 and organic compounds. These growth processes are generally limited to particles less than 1 μm in diameter.

2.6 PARTICLE TRANSPORT AND REMOVAL

Mechanisms for particle transport and removal are dictated by particle size. Coarse particles are typically emitted close to the surface and are quickly removed through gravitational effects (sedimentation) or inertial effects (impaction).

For particles less than 1 μm in diameter, sedimentation is not an effective means of removal. Instead, the main removal mechanisms are:

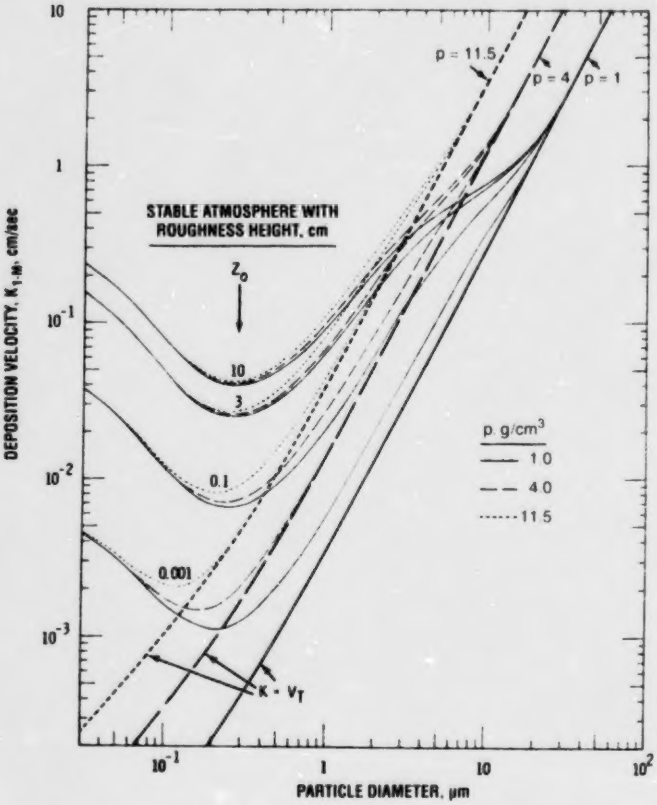
(a) dry deposition – the removal of chemical species or particles from the atmosphere at the air-surface interface, and

(b) precipitation scavenging – the removal of chemical species or particles from the atmosphere by interaction with precipitation.

Dry deposition is typically expressed in terms of the dry deposition velocity v_d. v_d is the downward flux, F, of a chemical species or particle divided by the concentration of that species $c(x, y, z_1, t)$ at some height z_1 above the surface (typically 1-2 m). Dry deposition can be viewed as a multistage process involving: (1) turbulent transport of a chemical species or particle from the boundary layer to the surface laminar sublayer; (2) diffusion across the surface laminar sublayer to the surface; and (3) uptake by the surface (Seinfeld, 1986). Factors that influence transport to the surface laminar sublayer include wind speed, atmospheric stability and surface roughness. Transport to the surface depends on molecular properties affecting diffusion across the laminar sublayer. Surface uptake may be assumed to be immediate once contact is made, although surface properties such as moisture content and canopy structure will affect resuspension (Sehmel, 1980). Superimposed on the rate of deposition is the particle settling velocity due to gravitational effects. Deposition velocities predicted by Sehmel (1980) for a stable atmosphere as a function of particle diameter, aerodynamic surface roughness (z_0) and particle density (ρ) are presented in Figure 2.4. Clearly, the lowest deposition velocities are predicted for particles in the accumulation mode (i.e., 0.1-1.0 μm). Sehmel (1980) notes that predictions by Sehmel and Hodgson (1978) for an unstable atmosphere show increased deposition velocities relative to those for a stable atmosphere. However, this increase is small relative to the effects of particle diameter, friction velocity and surface roughness. Given the many factors involved in the characterization of dry deposition, estimating deposition rates is an onerous task. For a more detailed review of the dry deposition process, the reader is referred to Sehmel (1980) or Seinfeld (1986).

Removal through precipitation scavenging may take place through in-cloud or below-cloud processes. "Rain-out" is an in-cloud process in which particles serve as cloud condensation nuclei (CCN) or are captured in the formation of cloud droplets. To act as CCN at 1% supersaturation, the particles must be at least 0.1 μm in radius if wettable but water-insoluble and at least 0.01 μm in radius if water soluble. Forest fires are thought to be major sources of CCN, whereas windblown dust and sea salt particles are considered minor sources. Sulphates have been identified as a common constituent of CCN. "Wash-out" is a below-cloud process in which particles are scavenged by falling precipitation. Particles greater than 2 μm in diameter are the most efficiently removed by this process. Globally, it is estimated that precipitation scavenging accounts for about 80-90% of the mass of particles removed from the atmosphere (Wallace & Hobbs, 1977). Improvements in visibility following precipitation events are generally attributed to precipitation scavenging. For a more detailed discussion of scavenging processes, the reader is referred to US EPA (1982a).

Figure 2.4 Predicted Deposition Velocities at 1 m for μ=20 cm/s and Particle Densities of 1, 4 and 11.5 g/cm^3. V_T is terminal settling velocity (Sehmel, 1980).



2.7 CHEMICAL CHARACTERISTICS

In the preceding sections, the formation and removal processes affecting the fine and coarse fractions of particulate matter have been discussed and have been shown to be distinctly different. It is not surprising, therefore, that the chemical species found within the fine and coarse fractions should also vary significantly.

A schematic presenting an idealized aerosol mass distribution and the typical segmentation of chemical species among the fine and coarse fractions is shown in Figure 2.5. The major components of the fine fraction include sulphate, nitrate, ammonium, lead and inorganic and organic carbon compounds. These particles are generally of primary anthropogenic or secondary origin, although forest fires are known to be a large natural source of particulate matter in the fine fraction. Particles in this size range tend to be acidic in nature.

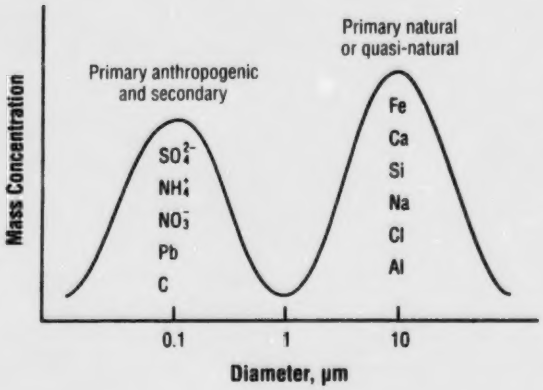
The coarse fraction consists primarily of materials that are common to the earth's crust (oxides of iron, calcium, silicon and aluminum) and sea salt spray (sodium and chloride). Particles of this size range are typically basic in nature.

Some chemical species such as nitrates and chlorides may be found in both the fine and coarse fractions. Trace metals such as nickel, tin, vanadium, copper, zinc, antimony and manganese are also variable. The following sections provide a detailed description of the more common chemical species associated with PM.

2.7.1 Elemental Carbon

Inorganic or elemental carbon (EC), also known as graphitic or black carbon, is a product of the incomplete combustion of carbon-based materials and fuels, and is solely primary in origin. The predominant sources of EC include fossil fuel combustion and biomass burning (Penner et al., 1993).

Figure 2.5 Idealized Volume (mass) Distribution of Fine and Coarse Particles in an Urban Atmosphere, and Associated Chemical Composition (from Seinfeld, 1986)



EC particles are typically less than 1 μm in diameter. Size distribution measurements of EC in ambient air and vehicular emissions have been found to exhibit a bimodal distribution with peaks in the range of 0.05-0.12 μm and 0.5-1.0 μm (Venkataraman and Friedlander, 1994). Because EC particles are both very small and very inert, they can remain in the atmosphere for extended periods before being removed by scavenging processes. This poses a potential health problem, as these particles can penetrate deeply into the lungs and act as adsorption sites for toxic pollutants. A characteristically large surface area together with its impurities makes EC the principal light-absorbing aerosol species, with the potential to affect both visibility and climate (Charlock and Sellers, 1980; Charlson et al., 1992). It is estimated that EC is responsible for more than 90% of light absorption and 25-45% of overall visibility reduction (Hamilton and Mansfield, 1991), as described in chapter seven.

2.7.2 Organic Carbon

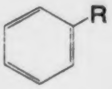
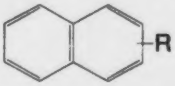
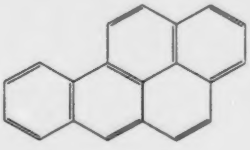
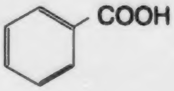
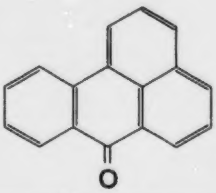
Organic carbon (OC) particulate matter can be directly released to the atmosphere or produced via secondary gas-to-particle conversion processes. It is predominantly found in the fine fraction (Van Vaeck and Van Cauwenberghe, 1978). Surprisingly, meat-cooking operations, paved road dust and fireplaces have been estimated to account for more than 50% of fine OC particle emissions in an urbanized core of the Los Angeles Basin (Hildemann et al., 1994). The associated organic species have been found to number in the hundreds (Rogge et al., 1991, 1993a, 1993b, 1993c). The major classes of OC compounds found in urban particulate matter are summarized in Figure 2.6. Alkanes in the range of C_{17}-C_{36}, alkenes, and aromatic and polycyclic aromatic hydrocarbons (PAH) are typically primary in nature (Seinfeld, 1986). Oxidized hydrocarbons such as acids, aldehydes, ketones, quinones, phenols and esters may be primary or secondary in nature. Nitro derivatives of organic compounds and heterocyclic sulphur compounds have also been identified in organic particulate matter.

PAH compounds are some of the most well-studied of the organic compounds, as certain species such as benzo[a]pyrene have been identified as potential carcinogens. Rogge et al. (1991, 1993a, 1993b, 1993c) identified PAH compounds in particulate emissions from vehicle exhaust, road dust, charbroiler and meat cooking operations and natural gas home appliances. Ambient measurements in Barcelona, Spain (Aceves and Grimalt, 1993) revealed that airborne PAH levels measured in very fine (<0.5 μm) particulate matter were more than fifty times higher than those levels found in coarse (>7.2 μm) particles. Measurements of PAH in ambient air and vehicular emissions were found to exhibit bimodal distributions similar to that for EC, with peaks in the range of 0.05-0.12 μm and 0.5-1.0 μm (Venkataraman and Friedlander, 1994). The more volatile four-ring PAHs such as fluoranthene, pyrene, benz[a]anthracene and chrysene were found to predominate in the nuclei mode particles while five-ring and larger particles such as benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, dibenz[a,h]anthracene, benzo[ghi]perylene and indeno[1,2,3-cd]pyrene predominated in the accumulation mode particles.

Increasing interest has focussed on secondary organic compounds (e.g. Grosjean and Seinfeld, 1989; Grosjean, 1992; Pandis, et al., 1992, 1993), as they are estimated to form a substantial portion of the fine fraction of PM_{10}, with contributions of up to 50% estimated in parts of the Lower Fraser Valley in British Columbia (Lowenthal et al., 1994) and 26-47% estimated for selected areas in the United States (van Houdt, 1990). At the height of a pollutant episode studied in the Los Angeles Basin, secondary formation was estimated to account for approximately 70% of organic particulate matter (Turpin and Huntzicker, 1991).

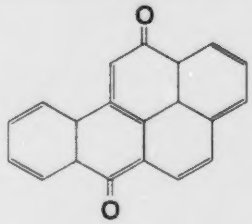
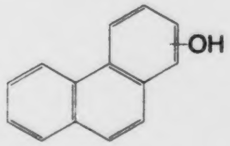
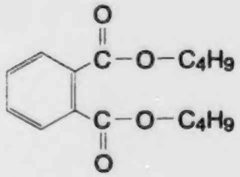
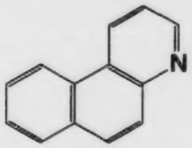
Secondary processes involve the gas-phase oxidation of hydrocarbon precursors by the hydroxyl radical during the day, nitrate radicals during the night, and ozone (O_3) during the day or night (Grosjean, 1992). Based on an examination of the rate constants and of the relative concentrations of electrophiles in urban air, aromatic hydrocarbons, saturated aliphatics (e.g., alkanes, cycloalkanes) and oxygenated aliphatics (e.g., alcohols, esters) are expected to react with the hydroxyl radical. Likewise, alkenes, cycloalkenes and other olefinic compounds such as dienes, terpenes and styrene are expected to react with the hydroxyl and nitrate radicals and ozone. Low-volatility products either nucleate or condense on the surfaces of preexisting particles, yielding particles in the size range of 0.1-1.0 µm. Compounds which yield high-volatility products will be unable to form particulate matter under atmospheric conditions. Such compounds include the C_1-C_6 alkanes, the C_2-C_6 alkenes, benzene and many low-molecular-weight carbonyls, chlorinated compounds and oxygenated solvents (Grosjean and Seinfeld, 1989).

Figure 2.6 Major Classes of Organic Compounds Found In Urban Air (from Daisey, 1980)

Compound Class	Example	Concentration in Urban Air (ng m^{-3})	Location, Date of Measurement(s)
Alkanes	n-$C_{22}H_{46}$	1000-4000	217 U.S. urban stations 1966-67
Alkenes	n-$C_{22}H_{44}$	2000	217 U.S. urban stations
Alkylbenzenes		80-680	West Covina, California July 24, 1973
Naphthalenes		400-500	Pasadena, California September, 1972
Polycyclic aromatic hydrocarbons		6.6	100 U.S. urban stations, 1958-59
		3.2	32 U.S. urban stations, 1986-87
	(Benzo (a) pyrene)	2.1	32 U.S. urban stations 1970
Aromatic acids		90-380	Pasadena, California September 1972
		8	Average U.S. urban prior to 1965
Cyclic ketones		2-48	U.S. urban site January 1968

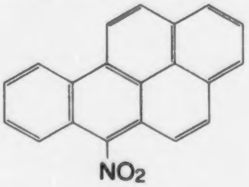
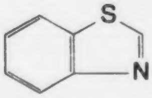
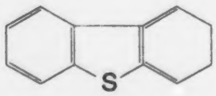
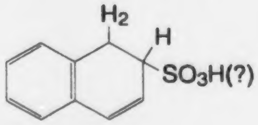
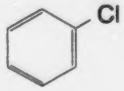
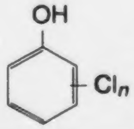
(continued)

Figure 2.6 Major Classes of Organic Compounds Found in Urban Air *(continued)*

Compound Class	Example	Concentration in Urban Air (ng m^{-3})	Location, Date of Measurement(s)
Quinones		0.04-0.12	Toronto, Ontario 1972-1973
Phenols		~ 0.3	Antwerp, Belgium 1975
Esters		29-132	Antwerp, Belgium 1976
		2-11	New York City 1975
Aldehydes	$CHO(CH_2)_nCHO$	30-540	Pasadena, California September 1972
Aliphatic carboxylic acid	$C_{15}H_{31}COOH$	220	New York City February 1964
		36.5	Antwerp, Belgium January 1976
Aliphatic dicarboxylic acids	$HOOC(CH_2)_nCOOH$	40-1350	Pasadena, California September 1972
Aza-arenes		0.2	Composite 100 U.S. urban sites, 1963
		0.01	New York City 1976
		~ 0.5	Antwerp, Belgium
N-Nitrosamines	$(CH_3)_2NNO$	≤ 0.03-0.96	Baltimore, Maryland August 1975
		15.6	New York City July 1976
Nitro compounds	$CHO(CH_2)_nCH_2ONO_2$	40-1010	Pasadena, California September 1972

(continued)

Figure 2.6 Major Classes of Organic Compounds Found In Urban Air *(continued)*

Compound Class	Example	Concentration in Urban Air (ng m^{-3})	Location, Date of Measurement(s)
Nitro compounds		identified	Prague, Czechoslovakia
Sulfur heterocyclic compounds		0.014-0.02	New York City 1976
		identified	Indianapolis and Gary, Indiana
SO_2-adducts		2-18 nmoles m^{-3}	New York City 1976
Alkylhalides	$C_{18}H_{37}Cl$	~ 20-320	Pasadena, California September 1972
Arylhalides		0.5-3	Pasadena, California 1972
Chlorophenols		5,7-7.8	Antwerp, Belgium 1976

2.7.3 Trace Elements

Highly volatile trace elements such as lead, nickel, arsenic, chromium and cadmium are emitted in vapour form from combustion and other high-temperature industrial processes and quickly condense in the ambient atmosphere. These species are known to concentrate in the fine fraction (Fennelly, 1975). Oxides of silicon, aluminum, iron, potassium, calcium and magnesium are common constituents of the earth's crust and common constituents of the coarse fraction. Sodium from sea spray is also commonly found in the coarse fraction.

Where trace elements exhibit a high abundance relative to other elements in a source profile, they may be used as key-indicating species for that source. Vanadium is a common tracer for heavy oil combustion, whereas zinc is used to identify municipal refuse incinerators (Cheng and Hopke, 1989; Schroeder et al., 1987). Iron is a marker for suspended crustal material (Lyons et al., 1993). Selenium is associated with coal combustion products (Andren et al., 1975). Lead is still used to identify motor vehicles, although with the phase-out of leaded gasoline alternative markers such as PAH compounds are being investigated (Li and Kamens, 1993).

In a review of elemental size distributions for 38 trace metals reported in the literature over the preceding two decades (based exclusively on cascade impactor data), elements with a MMAD of less than 2 μm were found to exhibit bimodal distributions, with a dominant peak in the range of 0.5-1 μm and a secondary peak at 3-5 μm (Milford and Davidson, 1985). This included lead, mercury, selenium, cadmium, antimony, bromine, nickel, arsenic, chromium, zinc and copper. Elements with a MMAD greater than 2 μm exhibited unimodal mass distributions, with the dominant peak at 3-5 μm and little mass below 1 μm in diameter. This included manganese, cobalt, chlorine, iron, potassium, sodium, silicon, aluminum, calcium, magnesium and strontium. More recent measurements made in the Los Angeles Basin reveal trimodal distributions for lead and manganese, with the smallest mode attributed to fresh emissions from motor vehicles and supermicrometre particles attributed to suspended crustal material (Lyons et al., 1993). An intermediate mode (0.5-1.0 μm) was believed to result from the growth of smaller particles.

2.7.4 Sulphate

Sulphate is one of the predominant species in the fine mode. Particle sulphate species found in the atmosphere include the strong acids H_2SO_4 and NH_4HSO_4 and the weak acid $(NH_4)_2SO_4$ (Lipfert et al., 1989). In the eastern United States, where SO_2 emissions from coal- and heavy-oil-fired plants are substantial, it has been estimated that sulphates, associated ammonium ion and water comprise approximately 40-70% of the fine particle mass. These contribute substantially to regional haze problems (Husar and Wilson, 1993). Sulphate contributions of 65% have been observed in southwestern Ontario in the summer (Keeler et al., 1990). Measurements taken during the Canadian Acid Aerosol Measurement Program (CAAMP) study in eastern Canada during 1992-1993 exhibited average sulphate to $PM_{2.5}$ ratios of 0.39, 0.30, 0.35, 0.30, 0.24, 0.36 and 0.27 at Egbert, Kejimkujik, Sutton, Windsor, Montréal, Saint John and Toronto, respectively (Environment Canada, 1994). On average, 32% of the $PM_{2.5}$ was due to sulphate, and 60% of the PM_{10} was due to $PM_{2.5}$. Nationally, composite average sulphate concentrations observed at sites east of Winnipeg are 2.3 times higher than those observed at western sites (Dann, 1994). The regional nature of particulate episodes in eastern Canada is attributed to the long atmospheric residence times of sulphate and the synoptic scale of the meteorological conditions conducive to episode development.

A detailed description of particle sulphate formation mechanisms is found in US EPA (1982a). Briefly, sulphate may be formed through the oxidation of SO_2 in the gas phase or aqueous phase. Gas-phase oxidation is primarily via reaction with the hydroxyl radical (Atkinson and Lloyd, 1984) to form sulphuric acid gas. Because of its low vapour pressure, sulphuric acid gas may nucleate in the presence of water vapour to form sulphuric acid droplets. Sulphuric acid gas may also condense on existing particles or react with ammonia to form the progressively neutralized compounds $(NH_4)HSO_4$ and $(NH_4)_2SO_4$. Calvert and Stockwell (1983) reported a wide range of transformation rates (0.01-5 %/hr of SO_2 concentration), with nighttime rates less than 0.1 %/hr. Their studies indicate that gas-to-particle transformation is controlled by the hydroxyl radical and competing reactions as opposed to SO_2 concentrations. Hydroxyl radical production is related closely to photochemistry. Therefore, the highest rates of transformation are expected to occur during the summer daytime when photochemistry is most active.

Aqueous-phase oxidation by oxygen and hydrogen peroxide is a second important pathway for sulphate production (Saxena and Seigneur, 1987). Reaction with oxygen is dependent on iron and manganese concentrations and on liquid water content, which, in turn, is dependent on relative humidity and background concentrations of sulphate, nitrate and ammonia (Saxena et al., 1986). Reaction with hydrogen peroxide is important at low temperatures when gas solubilities are relatively high. In the presence of fog or clouds, SO_2 can dissolve in droplets, where it undergoes faster aqueous reactions than in the gas phase. In the presence of ammonia, H_2SO_4 is neutralized to form $(NH_4)_2SO_4$. Reactions within the fog droplet are controlled by the solubility of precursor gases, and are generally 10-100 times faster than gas-phase reactions (Watson et al., 1994a).

The size distribution for sulphate particles is associated with the accumulation mode, (Milford and Davidson, 1987). Particles produced through gaseous-phase oxidation are typically smaller (MMAD approx. 0.2 μm) than those produced through aqueous-phase oxidation (MMAD approx. 0.5 μm) (Hering and Friedlander, 1982). This feature is used to study the relative importance of the two formation pathways. The presence of supermicrometre particles in marine environments may be attributed to sea spray (MacIntyre, 1974) or the oxidation of SO_2 originating from biogenically produced dimethylsulphide or anthropogenic sources (Garland, 1981). In continental locations, scanning electron microscopy studies revealed sulphate particles attached to clay and calcite particles and smooth sulphate coatings on clay, calcite and pollen particles, suggesting gas-to-particle conversion (Mamane and Noll, 1985).

MMADs averaged over a number of marine and continental distributions for particle sulphate were calculated to be 2.3 and 0.52 µm, respectively (Milford and Davidson, 1987). This compares with more recent estimates of 0.25-0.31 µm in the continental United States (Bondietti and Papastefanou, 1993).

Sulphate particles may be transported long distances (Pierson et al., 1990; Tanner et al., 1984). For a particle of 0.2 µm MMAD, an atmospheric residence time of 12 days is estimated (Bondietti and Pastefanou, 1993). Usually a residence time of 3-5 days is expected for sulphate particles (US EPA, 1996).

2.7.5 Nitrate

Particle nitrate is an end-product of photochemical reactions involving nitrogen oxides. It has been identified as a major constituent of secondary particulate matter in urban areas in British Columbia (Pryor and Steyn, 1994a) and the western United States (Gray et al., 1988; Chow et al., 1992a, 1992b) and a key contributor to visibility problems in the Lower Fraser Valley (Pryor and Steyn, 1994b), Denver (Groblicki et al., 1981) and Los Angeles (White and Roberts, 1977).

Particle nitrate is formed by reactions involving nitric acid (HNO_3) and either ammonia or preexisting particles. Nitric acid is formed primarily by gas-phase reactions of NO_2 with the hydroxyl radical. Additional HNO_3 is produced at night during the hydrolysis of dinitrogen pentoxide (N_2O_5), which is a by-product of reactions involving NO_2 and the nitrate radical (NO_3). This pathway is ineffective during the daylight hours due to the reactivity of both N_2O_5 and NO_3. Gas-to-particle transformation rates for NO_2 to HNO_3 are rapid compared with those for sulphates, ranging from 1%/hr to 90%/hr (Calvert and Stockwell, 1983). These rates are significant during both the daytime and nighttime, in contrast to gas-to-particle sulphate production which is most active during the daytime. Aqueous-phase reactions involving HNO_3 or NH_4NO_3 are an additional pathway for particle nitrate formation. Studies have shown that in the presence of sufficient ammonia, equilibrium is established between gaseous HNO_3, ammonia and solid-phase ammonium nitrate (NH_4NO_3) (Stelson, 1979; Stelson and Seinfeld, 1982a, 1982b). This equilibrium is strongly influenced by temperature and relative humidity, with particle formation being favoured by low temperatures and higher relative humidities (Russell et al., 1983). However, more recent studies (Wexler and Seinfeld, 1990) have shown that the equilibrium assumption may not hold for lower temperatures or larger particles when the time scales for mass transport are too large. Ammonium nitrate is characterized by high vapour pressures. As a result, ammonia will distill away from NH_4NO_3 in the presence of acidic sites such as less than fully neutralized acidic sulphates.

A further pathway for nitrate formation involves reactions that convert sodium chloride to hydrochloric acid and thermally stable nitrates such as sodium nitrate (Russell and Cass, 1984; Mamane and Mehler, 1987; Pilinis and Seinfeld, 1987; Pio and Harrison, 1987). This mechanism is believed to be the principal source of coarse particle nitrate and is a factor where atmospheric sodium chloride levels are significant. Major sources of sodium chloride include sea salt, road sanding and dry lake beds.

The MMAD for particle nitrate distributions has been reported to range from 0.23-4.2 µm (Milford and Davidson, 1987). However, volatility losses and sampling artifacts may render this data unreliable.

2.8 SUMMARY

PM_{10} refers to all airborne solid and liquid particles, except pure water, that are 10 µm or less in diameter. PM_{10} is generally subdivided into a fine fraction of 2.5 µm or less ($PM_{2.5}$) and a coarse fraction of larger than 2.5 µm, reflecting its multimodal nature. It is further classified as primary (emitted directly into the atmosphere) or secondary (formed in the atmosphere through chemical and physical transformations). Primary particles are found in both the fine and coarse modes, whereas secondary particles such as sulphates and nitrates are found predominantly in the fine fraction.

The behaviour of PM_{10} in the atmosphere is affected by many chemical and physical parameters, including particle size. The theory behind particle size distributions and observations taken from various environments were presented. Particles less than

0.1 μm in diameter (the nuclei mode) are formed primarily from the condensation of hot vapours during combustion processes and from the nucleation of atmospheric species to form new particles. Although the greatest number of airborne particles are found in the nuclei mode, they contribute little to overall particle mass loading. They are subject to Brownian motion and coagulation processes that quickly yield larger particles. Particles in the size range of 0.1-2.0 μm (the accumulation mode) result from the coagulation of particles in the nuclei mode and from the condensation of vapours onto existing particles that then grow into this range. They account for most of the particle surface area and much of the particle mass. The accumulation mode is so-named because removal processes are least efficient in this size range. Particles can remain in the atmosphere for days to weeks. Particles larger than 2.0 μm (the sedimentation or coarse mode) are typically associated with mechanical processes such as wind erosion, industrial processes that produce fly ash, and breaking ocean waves that produce sea spray. These particles are efficiently removed by gravitational settling and remain in the atmosphere for periods of a few hours to a few days. They contribute little to number concentrations but significantly to total particle mass.

Other physical characteristics that affect particle behaviour include particle shape and density, bulk properties such as chemical composition, vapour pressure, hygroscopicity, deliquescence and refractive index, and surface properties such as electrostatic charging and surface films. Deliquescence is of particular interest. As deliquescent materials (e.g., ammonium bisulphate, ammonium sulphate and ammonium nitrate) will grow rapidly when the relative humidity rises above the material's deliquescence point, into a size range that efficiently scatters light and thus contributes to visibility reduction.

The two basic processes involved in particle formation were discussed: grinding or atomization, and the nucleation of supersaturated vapours. Windblown dust and sea spray are produced by the former process, whereas sulphate formation is an example of the latter process. Particle growth may occur through particle-particle interactions such as coagulation, or gas-particle interactions such as the absorption and adsorption of pollutant gases such as sulphur dioxide followed by chemical reactions to yield particulate sulphate. The main removal mechanisms are dry deposition, which involves the removal of particles from the atmosphere at the air-surface interface, and precipitation scavenging, which involves the removal of particles from the atmosphere by interaction with precipitation.

The chemical composition of PM_{10} generally varies between the fine and coarse modes. Usually, the fine mode is characterized by chemical constituents such as sulphate, nitrate, ammonium, lead, elemental carbon, metals, and hundreds of different organic carbon compounds. The coarse mode is characterized by materials typical of the earth's crust (oxides of iron, calcium, silicon and aluminum) and sea spray (sodium and chloride). Sulphates, nitrates, ammonium and a portion of the organic carbon compounds result from secondary reactions, while the remaining materials are typically primary in nature. Size distributions associated with various materials were described.

3 SAMPLING OF PARTICULATE MATTER

Measurements related to current compliance monitoring are expressed in terms of mass. Mass measurements may be made directly or indirectly. Direct (or manual) measurements are made by collecting particles on a pre-weighed filter over a specified period, weighing the soiled filter, and then dividing the gain in mass by the volume of air sampled. Samples are typically collected for a 24 h period, once every six days, as in the National Air Pollution Surveillance (NAPS) network (Dann, 1994). However, different sampling periods and frequencies may be used where required. Examples of manual samplers include high-volume (hi-vol) and dichotomous (dichot) samplers.

Indirect measurements are made using techniques other than gravimetric ones to assess particle properties. These measurements are then related to mass. Tapered element oscillation microbalance (TEOM) and beta-attenuation monitors are examples of indirect methods that have also been automated to provide near real-time measurements of particulate concentrations.

In this chapter, measurement techniques used in Canada and elsewhere are described and compared. Section 3.1 describes direct measurement techniques for TSP, PM_{10} and smaller particulate size fractions, filter media, mass measurements and chemical analyses. Section 3.2 reviews indirect measurement techniques for PM_{10} and its sub-fractions. Section 3.3 describes specialized sampling systems used to provide a more detailed chemical characterization of particulate matter. Personal Exposure Monitors (PEMs) are briefly reviewed in Section 3.4. Siting criteria are discussed in Section 3.5. A summary is provided in Section 3.6.

3.1 DIRECT MEASUREMENTS

For historical reasons, this section begins with a description of the standard technique for total suspended particulate (TSP) measurements even though the document focuses on the smaller size fractions of particulate matter. This is followed by a description of techniques available for PM_{10} measurement: the size-selective inlet (SSI) hi-vol sampler, the dichot sampler and the Partisol sampler. Additional techniques to measure particle fractions smaller than 10 µm are also described. Finally, filter media, mass measurements and chemical analyses are briefly discussed.

3.1.1 TSP

The standard sampler for TSP monitoring is the hi-vol sampler. The hi-vol sampler draws large volumes of air (1132 L/min or 40 cfm) through a 20.3 x 25.4 cm filter. TSP concentrations are calculated based on the net weight gain of the filter and the volume of sampled air. The collected particulate sample may be further analysed for its chemical constituents.

A characteristic feature of the hi-vol sampler is the gabled roof, designed to prevent dustfall on the filter. Because of this design, particle collection efficiency is dependent on both wind speed and wind direction. The 50% cut point (D_{50}) is the particle aerodynamic diameter at which 50% of the particles pass through the inlet and 50% are rejected. The hi-vol sampler has a poorly defined cut point which has been shown to decrease from 50 µm in 2 km/h winds to about 22 µm in 24 km/h winds (McFarland and Rodes, 1979). Positive biases of 10-15% can occur due to passive deposition onto the filter (Bruckmen and Rubino, 1976; Chahal and Romano, 1978; Swinford, 1980).

3.1.2 PM_{10}

To fractionate the sample into specified size ranges such as for PM_{10}, specially designed sampling inlets are used. PM_{10} samplers are designed to have a 50% cut point of 10 µm. The cut point is valid for a specified flowrate, and will change with the inverse square root of the ratio of actual flow to cut point flow.

As shown in Table 3.1, sampler inlets may vary as to the method of fractionation. Cyclonic flow, inertial impaction and virtual impaction are commonly applied separation techniques that are described later in this chapter. Also evident from Table 3.1 is that the 50% cut point varies slightly among sampler models. The

Table 3.1 US EPA-approved Reference and Equivalent Methods for PM_{10} (based on Chow, 1995)

Manufacturer	Operating Principle	d_{50} (µm)	Flow Rate (L/min)	PM_{10} Reference Number	Comments
SA or GMW Model 321A	Impactor	10.2	1,133	RFPS-1287-065	2-stage with greased shim
SA or GMW Model 321B	Impactor	9.7	1,133	RFPS-1287-064	2-stage with greased shim
SA or GMW Model 1200	Impactor	9.7	1,133	RFPS-1287-063	Single-stage with greased shim (body hinged)
Wedding PM_{10}	Cyclonic Flow	9.6	1,133	RFPS-1087-062	Inlet cleaning port on top of inlet
SA 254 Medium Volume PM_{10} Inlet	Impactor	10	113	RFPS-0387-071, EQPM-0990-076	Several small screws must be removed to clean.
SA 246B	Virtual Impactor	10.2	16.7	EQPM-0391-081	Plastic cap acts as a hopper to collect large particles

resulting concentration differences due to cut point biases may be amplified because the 10 µm cut point occurs near a maximum in the particle mass distribution.

Dichotomous Sampler

The dichot sampler was the original sampler for inhalable particles. It is modelled after inertial virtual impactors in that it fractionates particles according to their aerodynamic characteristics. The dichot sampler operates at a low sampling rate (16.7 L/min) relative to the hi-vol sampler. Sample air is drawn through an inlet head, and then through a virtual impactor that separates particles into a coarse (>2.5-10 µm) and fine (2.5 µm) fraction. This is achieved by accelerating particles through a nozzle and then drawing 90% of the flow stream off at right angles. The fine particles follow the right-angle flow stream, while the larger particles continue toward the collection nozzle. Particles are collected on 37 mm filters. Separation of the two size fractions minimizes potential interactions between the more acidic fine fraction and the more basic coarse fraction. However, this also requires further care in sample handling and mass determination than required with the SSI samplers. As well, it requires different mass and analytical techniques than used for the hi-vol filters.

Size Selective Inlet High-Volume Sampler

Reflecting the need to monitor inhalable particles, the US EPA commissioned the design of an SSI to retrofit standard hi-vol samplers. The SSI head is round-shaped to allow for unbiased sampling from any direction, in direct contrast to the gabled roof design of the original hi-vol sampler (Hicks and Corr, 1983). Air is drawn through a series of baffles to separate out particles greater than 10 µm in diameter. Similar to the standard hi-vol samplers, units are operated at a flow rate of 1132 L/min (40 cfm) and samples are collected on 20.3 x 25.4 cm fibre filters. The same mass and analytical techniques as used for the TSP filters can be used for the filters from the SSI samplers. A potential disadvantage with respect to these samplers is the self-contamination of the filter through wear of the carbon/graphite brushes against the copper segments of the motor commutator that are part of the internal electrical mechanism.

Partisol Sampler

The Partisol sampler is a manual, low-volume sampler approved by the US EPA as a reference method for PM_{10} (US EPA, 1994a). It consists of a hub station and up to three satellite stations. The hub unit contains a microprocessor with internal data storage, an active flow control system and a pump. All satellite units are fitted with a sample inlet and a 47 mm filter exchange mechanism that may employ quartz fibre, Teflon-coated glass fibre or Teflon filters. Only one unit can operate at a time. Menu-driven software can be used to determine the conditions (e.g., wind speed, wind direction) under which the hub or satellite units are active. It may be fitted with TSP, PM_{10}, $PM_{2.5}$ or PM_{10} inlet heads. Therefore, compared with other manual samplers, it provides added flexibility with respect to sampling inlet, sample collection hardware and pre-programmed operation. Flow is controlled between 3-20 L/min.

Comparability of Direct PM_{10} Measurements

An intercomparison study of the Sierra Andersen (S/A) 321A PM_{10} sampler and the Wedding PM_{10} monitor was conducted by Purdue et al. (1986) in Phoenix, Arizona. Unoiled S/A 321A samplers were found to collect an average of 58% more mass than co-located and uncleaned Wedding samplers. Part of the difference was attributed to cut point bias. However, during extended sampling periods when sampling performance was degraded, the S/A 321A monitor tended to over-sample, while the Wedding monitor tended to under-sample. By periodically cleaning the inlets and oiling the impaction plates of the S/A 321A, differences were reduced to within predictable agreement based on expected mass calculations from wind tunnel inlet effectiveness data and ambient particle mass distribution data.

An intercomparison of various PM_{10} monitors (Wedding, S/A 321B, S/A 1200 and S/A dichot) was carried out in Rillito, AZ during a year-long measurement program (Thanukos et al., 1992). The S/A and Wedding PM_{10} sampler inlets differ in the type of fractionation device used. S/A inlets incorporate a greased impaction surface below a series of impactor jets, while Wedding inlets employ a greased cyclone. Comparison of concurrent measurements by different samplers showed very linearly but significant differences in measured concentrations when all instruments are consistently maintained. In summary, the Wedding samplers recorded the lowest concentrations, followed by the S/A 321B samplers (19.2% higher than the Wedding samplers), the S/A 1200 samplers (28.1% higher than the Wedding samplers), and the S/A dichot samplers (41.6% higher than the Wedding samplers).

In the Canadian national network, Graseby Andersen (formerly Sierra Andersen) dichot and Graseby Andersen SSI samplers are collocated at five sites: Saint John, Ottawa, Edmonton, Vancouver Rocky Point Park and Vancouver West 10th Ave (Dann, 1994). Before 1988, the S/A 321 inlet was used in all the SSI samplers. This inlet was shown to allow large particle pass-through from bounce and/or re-entrainment, resulting in an over-sampling of PM_{10} (Hayes et al., 1988). This was particularly problematic when coarse particle loadings were high. Consequently, the S/A 321 inlets were replaced by Graseby Andersen 1200 inlets in December 1988. Based on samples collected between 1984-1993, mean ratios of PM_{10} concentrations measured with the dichot samplers to PM_{10} concentrations measured with hi-vol samplers ranged from 0.93 at sites in Edmonton and Saint John to 1.17 at the Vancouver West 10th Avenue site (Dann, 1994). Excellent correlation was found between the samplers (0.84-0.95).

3.1.3 Fine Fraction

Similar operating principles used in PM_{10} inlets are applied to inlets designed to collect smaller particle size fractions. However, in contrast to the 10 μm cut point, the 2.5 μm cut point occurs near a minimum in the particle mass distribution, thereby minimizing differences between samplers with cut point biases. In the following, methods applied to the direct measurement of fine particle fractions are described.

Inertial Impactors

Inertial impactors are based on the following principle of operation. An airstream is drawn through a series of nozzles of jets and directed toward a flat impaction plate. The high inertia of larger particles will carry them into the impaction plate, which may be coated with an adhesive or grease to aid collection. Smaller particles will continue with the airstream.

The cascade impactor consists of a series of nozzles and coated impaction plates, called stages, with each stage collecting progressively smaller particles. Size segregation is dependent on sampler geometry and flowrate. Bounce-off, re-entrainment and deposition on lower stages, wall losses, and lack of discreteness are problems encountered with this technique. Sample collection is generally limited to particles of about 0.5 μm or larger, which eliminates a large percentage of urban particulate matter.

Virtual impactors differ from cascade impactors in that the impaction plate is replaced by an opening that directs larger particles to one sampling substrate, and the smaller particles to another. This principle is used in dichot samplers, as previously described. Relative to cascade impactors, the effects of particle bounce-off and re-entrainment are reduced. However, this method requires that part of the total flow (typically 10%) be drawn through the virtual impaction surface. Therefore, correction factors must be applied to the coarse channel flow to account for contamination by the fine fraction.

A low-flow (4 L/min) standard impactor was developed by Marple et al., (1987) for use in the Harvard Six City Study (Dockery and Spengler, 1981a,b). Also called the Harvard impactor, it was designed with 2.5 µm or 10 µm cut points and two identical cascade stages. The purpose of the latter is to produce sharper cut points and to reduce particle bounce resulting from particle overloading. The impactor surfaces are oil-impregnated metal plates to further reduce particle bounce. The Harvard impactor and variations on it have been used in several major indoor particulate monitoring studies: Total Human Environmental Exposure Study (THEES) (Buckley et al., 1988); Particle Total Exposure Assessment Methodology (P-TEAM) (Wallace et al., 1991); and New York State Energy Research Development Authority (ERDA) study (Koutrakis et al., 1992).

To obtain improved size fractionation of smaller particles, several low-pressure impactors have been developed: the Berner impactor (Berner and Lurzer, 1980); the Low Pressure Impactor (Hering et al., 1978; Hering and Friedlander, 1978); the Micro Orifice Uniform Deposit Impactor (MOUDI) (Marple et al., 1981); Model 20-725, Andersen Samplers Inc.; the DRUM (Davis Rotating-drum Universal-size-cut Monitoring impactor) sampler (Raabe et al., 1988). By operating under reduced pressure, the mean free path of the air becomes comparable to particle diameter, thereby reducing the drag on the particle and enhancing particle collection. Size segregation down to cut points of 0.025 µm (Berner and Lurzer, 1980) have been obtained. The Hering and Andersen samplers have very low flow rates of 1 and 3 L/min, respectively. As a result, sampling periods of more than 24 h may be required to collect sufficient sample (Bondietti and Papastefanou, 1993; Lyons et al., 1993).

Cyclone Samplers

Cyclone samplers use an impeller to impose a circular motion to air entering a cylindrical tube. Centripetal force moves the particles toward the walls of the tube. Particles reaching the walls, which may be covered in an oil or grease coating, either adhere to it or drop into a hopper at the bottom of the tube. The inner tube and hopper are cleaned to minimize re-entrainment. They are simple to operate, and can be very compact in design. As a result, cyclone samplers have been applied in a number of personal monitoring applications (e.g., Dockery and Spengler, 1981b; Spengler et al., 1985; and Colome et al. 1992). $PM_{2.5}$ cyclone inlets are also widely used in the IMPROVE monitoring network (Malm, 1994b).

Comparability of Direct $PM_{2.5}$ Measurements

Reflecting the past emphasis on PM_{10} as the particulate fraction of concern, relatively few intercomparison studies have been conducted for $PM_{2.5}$ samplers. To investigate potential sampling methodologies for the fine fraction, Cook et al. (1995) evaluated various $PM_{2.5}$ samplers in Bakersfield, California. Direct measurements were obtained from samplers that included two S/A 241 dichots, a Partisol 200H sampler, an AirMetrics Model 4.1 impactor and a Xontech 920 multi-channel sampler fitted with an AirMetrics 2.5 µm inlet. Indirect measurements (to be discussed more thoroughly in Section 3.3) were obtained with the TEOM Series 1400a monitor, Meteorology Research Inc.'s Model 1550 B nephelometer and the American Iron and Steel (AISI) tape sampler.

Good agreement was observed between the dichot samplers, the Partisol and the Xontech/AirMetrics sampler. Data pairs agreed within 10% of one another, and variability among samplers was less than 15%. The AirMetrics $PM_{2.5}$ unit did not yield good statistical agreement with the other samplers over the full range of particulate concentrations. Limited agreement was observed with the dichot data for concentrations of 20-50 mg/m^3. TEOM data collected at sampling temperatures of 30°C and 50°C were much lower than measurements from other samplers, suggesting volatilization problems due to the heating of the airstream. However, TEOM, AISI and nephelometer data correlated quite well to the other samplers at times, suggesting that these methods may be useful for monitoring $PM_{2.5}$ if strong and consistent relationships with the other samplers can be determined over time.

3.1.4 Filter Media

Filters consist of a woven mat or a permeable membrane that contains microscopic pores. Commonly used filter media for particle sampling are tabulated by Chow (1995), along with the references. No single filter type is suitable for all applications. Filter media used in monitoring activities must display the physical and chemical properties appropriate for the sampling methodology and the laboratory analysis methods used.

Selection should be based on the following considerations: particle sampling efficiency, mechanical stability, chemical stability, temperature stability, blank concentrations, flow resistance and loading capacity, cost and availability. Except for selective filtration, filters should remove more than 99% of the suspended particles drawn through them.

The two most common types of filters are fibre filters and membrane filters. Fibre filters exhibit a low pressure drop and high collection efficiencies for all particle sizes. Examples of fibre filters include those composed of glass fibres, Teflon-coated glass fibres and quartz material. Membrane filters retain particles on the surface of the filter, enabling non-depth analyses such as X-Ray Fluorescence. They generally have higher flow resistence and lower loading capacities than fibre filters, and are also more expensive. They are available in a wide range of materials including Teflon, Polyvinyl Chloride (PVC) and polycarbonate.

Filter materials exhibit varying degrees of reactivity with chemical species. Glass fibre filters adsorb HNO_3, NO_2, SO_2 and organic vapours, resulting in positive sampling artifacts. They are also characterized by high metal blanks. Quartz filters are relatively inert to the adsorption of gases, but passively adsorb organic vapours. Nylon filters exhibit high collection efficiencies for HNO_3. All filters may be subject to losses of volatile species due to changes in ambient temperature, pressure or relative humidity. Most nitrates are expected to be in the gas phase as HNO_3 at temperatures above 30°C, and in the particle phase at temperatures lower than 15°C (Russell et al., 1983). Significant losses (>50%) of nitrate, ammonium and chloride have been observed in glass and quartz fibre filters stored in unsealed containers at ambient temperatures for 2-4 weeks before analysis (Witz et al., 1990). Turpin et al. (1994), found that organic gas adsorption accounted for up to 50% of organic carbon on quartz fibre filters in southern California, and outweighed the amount of organic carbon lost to volatilization. This appears to be in contrast with the findings of Eatough et al. (1993), which showed that the percentage loss of semi-volatile compounds from a quartz filter during sampling accounted for approximately 50% (on average).

To minimize contamination of the filter by the operator, filter holders have been designed so that filters can be loaded and unloaded in a clean environment rather than in the field. Consideration should be given to the material used to construct the filter holder, particularly when measuring reactive components of the particulate matter. Additional consideration should be given to the filter holder configuration. Filter holders are designed as open-face or in-line (Chow, 1995). Open-face holders are without any constrictions upstream of the filter surface. In-line holders consist of a small diameter opening into a small chamber in which the filter is mounted. As a result, in-line filter holders may concentrate particles in the centre of the filter and will bias results where only part of the filter is analyzed. Differences of up to 600% have been reported between chemical measurements made at the middle and edges of filters sampled with in-line holders (Tombach et al., 1987; Fujita and Collins, 1989).

3.1.5 Mass Measurements

To determine particulate concentrations from manual samplers, mass measurement of the exposed filter is required. Minimum requirements for balance sensitivity are as follows: 100 mg for high-volume samplers, 10 mg for medium-volume samplers and 1 mg for low-volume samplers (US EPA, 1996). Measurements are made in a temperature- and humidity-controlled environment. The main interference results from electronic charges that induce non-gravimetric forces between the filter and the balance.

Most of the mass measurements for SSI and dichot samples obtained in the national monitoring network are done by the Pollution Measurement Division, Environmental Technology Centre (ETC) in Ottawa (Dann, 1994). A Mettler M3 or Mettler MT5 electronic microbalance is used. Filters are equilibrated at 23°C±3°C and 43%±5% prior to mass determination. The ETC laboratory uses a polonium 210 radioactive source to control static charge. Local agencies measure SSI samples collected from the Vancouver sites and three of the Saint John sites (Dann, 1994). Local agencies also measure most of the hi-vol filters. Lower detection limits of mass concentration are determined by the repeatability of filter tare weights. For SSI samples, the lower limit is about 2 $\mu g/m^3$. Corresponding limits for dichot samples are slightly lower. Upper limits are dependent on particle size. Small particles will clog the pores of the filter at low concentrations while larger particles will fall off the filter at high concentrations. For hi-vol samplers, the limit is typically in the range of 400-1000 $\mu g/m^3$ (Watson et al., 1989).

3.1.6 Chemical Analyses

Further analytical techniques may be applied to learn the chemical composition of particulate samples.

Various destructive and non-destructive methodologies are available. Chow (1995), lists some of these techniques, along with the associated references. The following discussion, therefore, is meant only to provide a brief introduction of these techniques.

X-ray fluorescence (XRF) and Proton Induced X-Ray Emission (PIXE) spectroscopy are two common techniques that can be used to determine concentrations of elements with atomic numbers from 11 (Na) to 92 (U). Both techniques leave the filter intact, enabling further analyses to be conducted on the filter. Neither requires sample preparation nor extensive operator time.

Inductively Coupled Plasma (ICP) emission spectroscopy and Instrumental Neutron Activation Analysis (INAA) are more expensive analyses than XRF or PIXE, however, they provide low detection limits for several species. INAA requires that the filter be wadded up and then made radioactive, while ICP destroys the filter.

Atomic Absorption Spectrophotometry (AAS) is useful for quantifying Na, Mg, K and Ca concentrations, but requires too great a dilution of the sample when multi-species analyses are required. Ion Chromatography (IC) and Automated Colorimetric (AC) analysis are applied to water extracts of filter samples to determine anion and cation content. IC is commonly used to measure chloride, nitrate and sulphate, while AC is commonly used to measure ammonium and chloride. Techniques developed to measure carbon species include Thermal/Optical Reflectance (TOR), Solvent Extraction Method and Thermal Combustion Method.

At the ETC laboratory, XRF is used for the elemental analysis of particulate samples (Dann, 1994). Lead analyses are conducted using a wavelength XRF spectrometer. Multi-element analyses are conducted using a Kevex 770/8000 energy dispersive X-ray fluorescence (EDXRF) spectrometer. Sulphate and nitrate analyses are done on the extracts of hi-vol and SSI filter cuttings using automated photometric methods. Cuttings are extracted in an ultrasonic bath (40 or 47 kHz) after addition of a wetting agent. Sulphate is determined using the methyl thymol blue method. Nitrate is determined as azo dye after reduction to nitrite.

3.2 INDIRECT MEASUREMENTS

As in Section 3.1, this discussion begins with a description of the historical methods for measuring particulate matter, such as the British Smoke Shade or Black Smoke (BS) sampler. This is followed by descriptions of the US EPA equivalent techniques for PM_{10} measurement (the beta attenuation monitor and the TEOM sampler) and the integrating nephelometer. Finally, a description of single particle samplers used to measure particle size distributions is provided.

3.2.1 Black Smoke Shade

The BS technique has been used in Britain since the early 1900s. Air is drawn at low flow rates (1.5 L/min) through a disc of cellulose fibre filter paper over a 24 h period. The reflectance of the resulting stain is analysed, yielding measurements in BS units. A calibration curve is then used to convert the units to particle mass concentration.

BS measurements are influenced by both the mass and optical properties of smoke. In particular, they are sensitive to the dark, "sooty" component of particulate matter in the size range of ≤3.5 to 4.5 µm. Therefore, BS measurements are generally considered a poor indicator of TSP levels. This technique is still being used in the United Kingdom and elsewhere in Europe because it is the main method for determining compliance with the European Community directive on suspended particulate, based on early epidemiological studies using BS data (EPAQS, 1995). The BS technique is also viewed as more accurately reflecting the health risks of smoke from coal combustion (Waller, 1980). It is also relatively simple and economical for routine sampling.

The commonly used relationship between BS measurements and gravimetrically determined concentrations were reported in a 1964 study supported by the Organization for Economic Cooperation and Development (OECD). Equivalent mass concentrations were determined through collocated gravimetric samplers. The US EPA (1982a) is careful to point out that the so-called OECD "high-volume" samplers are different from the US TSP hi-vol samplers. The OECD units were designed to be aerodynamically similar to the smoke shade unit, but had not been characterized for aerosol collection effectiveness. They operate at a sampling rate of 60 L/min, compared to approximately 1500 L/min used in the US model.

A non-linear relationship was found to exist between BS and TSP measurements made with co-located samplers (US EPA, 1982a). TSP measurements were generally 2 or more times higher than corresponding

BS readings, up to BS levels of about 100 μg/m^3. At higher levels, the two measurements tended to converge, with BS/TSP ratios of one at BS levels of 500 μg/m^3 or more. In a review of various European studies (Kretzschmar, 1975; Ball and Hume, 1977; Bailey and Clayton, 1982; Laskus, 1983; Edwards et al., 1983; Willison et al., 1985), Hamilton and Mansfield (1991) observed that background TSP concentrations were 2-4 times higher than the corresponding BS levels. At a roadside site, however, BS levels 3 times higher than the TSP levels were observed. This apparent anomaly was explained by the fact that the soiling factor for diesel-emitted smoke is 3 times higher than that for smoke from coal combustion, upon which the BS calibration chart is based.

3.2.2 Coefficient of Haze (CoH)

CoH is measured using the AISI tape sampler. The fundamental principles of the tape sampler are similar to those of the BS technique. Particles of up to approximately 5 μm in aerodynamic diameter are collected in a 25 mm spot on filter paper tape that is periodically (usually hourly) advanced to allow for collection of the next sample. The opacity of the stain is measured by the transmittance of light through the stained area. The result is expressed as optical density or CoH units per 1000 linear feet of air sampled. The CoH unit is 100 times the optical density of the deposit that allows 50% of light transmittance. The optical density is calculated as the log of the ratio of the light transmission through clean filter paper to that through the sample. Measurements can be made automatically, enabling hourly concentrations to be recorded.

The precision and accuracy of this method depends on the precision and accuracy of the air flow rate, filtration efficiency and optical system (Lodge, 1989). The lower limits of detection for optical density are about 0.05. Upper limits are about 0.30, which represents a 50% attenuation of the incident light.

Similar to BS measurements, CoH measurements are more representative of the fine, light-absorbing components of particulate matter such as soot particles. Conversion to units of mass therefore requires the use of a co-located gravimetric device. In a comparison of measurements from co-located CoH and dichot samplers in an urban Toronto location, a correlation of 0.75 was found between the $PM_{2.5}$ fraction and the CoH measurements, and one could not be interpolated from the other (Hicks and Corr, 1983). Fairley (1990) looked at the relationship between CoH and PM_{10} measurements in Santa Clara County from 1980 to 1986. The analysis was restricted to the winter months (November-January) of 1985 and 1986, when CoH levels were typically 3-4 times higher than in the summer months. Median CoH-to-PM_{10} ratios of 1.87 and 1.64 and corresponding correlation coefficients of 0.79 and 0.81 were reported for 1985 and 1986, respectively.

3.2.3 Beta Attenuation Monitor

The beta attenuation monitor, also called the beta-gauge monitor, has been used in Europe and Japan for several years. Mass determination is based on the attenuation that a beta-ray particle undergoes as it passes through an exposed filter. The beta attenuation monitor can provide hourly concentrations, based on a 55 min sampling period followed by calibration and measurement periods. The filter material is on a roll that can be automatically advanced. Therefore, compared with gravimetric techniques, filter handling is minimized, and therefore the potential for filter contamination reduced. Errors due to the loss of small fragments from the filter edges do not affect measurements; therefore, these monitors may be a better measure of lightly loaded samples than gravimetric techniques (US EPA, 1982a). The particle mass detection limit is about 5 μg/m^3 for a 1 h average.

A consideration in the use of beta attenuation monitors is that it requires a radioactive source. Further considerations are that the filter must be oriented in exactly the same way for each mass determination; changes in air density and atomic number of the collected particulate matter affect calibration; and the instrument is very expensive. Absorption of water by the filter material has been cited as a source of error (Lawrence Berkely Laboratory, 1975). The presence of a significant number of large particles has also been found to affect the precision and accuracy of measurements (US EPA, 1982a). Chemical characterization of the particulate matter requires the use of a co-located manual sampler.

Field comparisons of beta attenuation and hi-vol PM_{10} monitors have been done in Denver (Arnold et al., 1992) and in Citrus Heights, CA (Shimp, 1988). In the Denver study, a Wedding beta attenuation monitor reported 24 h average PM_{10} concentrations that were 19% lower than those measured by a Wedding hi-vol PM_{10} sampler. In the Citrus Heights study, the beta attenuation monitor averaged concentrations that were 6% less than collocated Sierra-Andersen Model 321a hi-vol PM_{10} samplers.

3.2.4 Tapered Element Oscillation Microbalance (TEOM)

In contrast to the previously described monitoring methods, the TEOM sampling method allows for the continuous measurement of PM_{10} concentrations. It has been approved as an equivalent PM_{10} monitoring method in the United States (US EPA, 1990), and is finding widespread application in various provinces (MELP, 1995; OME, 1995). It can also been fitted with a $PM_{2.5}$ sampling inlet (Rupprecht et al.,1995).

The TEOM instrument operates under the following principles. Ambient air is drawn in through a heated air inlet and an exchangeable filter cartridge that sits on the end of the tapered tube. The filtered air then goes through a hollowed tapered tube which is maintained in oscillation in a clamped-free mode. An automatic flow controller directed by the system's microprocessor maintains the sample flow at a constant rate. As particulate matter gathers on the filter cartridge, the tube's natural frequency of oscillation decreases. The electronics system continually monitors this frequency. Based upon the direct relationship between mass and frequency, the instrument's microprocessor computes the total mass accumulation on the filter, as well as the mass concentrations and mass rate, in real time. A more detailed description is contained in Patashnick and Rupprecht (1991).

Sample air is typically drawn in at a flow rate of 16.7 L/min, and a flow splitter channels a 3 L/min portion to be filtered. To standardize sampling and weighing conditions, the filter and the airstream passing through it are heated to 50°C, ensuring that the filter is always above the dew point and thereby minimizing water uptake.

In acceptance testing carried out in Birmingham, Alabama in 1990, three TEOM instruments were co-located with three reference PM_{10} hi-vol samplers (Meyer, 1993). The 24 h averages from each sampler correlated well (correlation coefficient of 0.977). The precision of replicate TEOM measurements (1 $\mu g/m^3$) was reported to be better than that of the reference samplers (1.8 $\mu g/m^3$). In testing done in Essen, Germany using two production version Series 1400a monitors, a minimum detection limit of 3.4 $\mu g/m^3$ was estimated for 1 h averages and 1.3 $\mu g/m^3$ for 24 h averages (RWTUV, 1994).

An evaluation of co-located TEOM and manual PM_{10} samplers by state and regional air agencies in the western United States showed that the TEOM data were consistently lower than that obtained by the manual samplers (Moore and Barthelmie, 1995). Mean percent differences ranged from -14% (all pairs greater than 20 $\mu g/m^3$) to -38% (all pairs greater than 150 $\mu g/m^3$). An analysis of approximately 1,000 pairs of TEOM-manual method data showed a -14% mean percent difference during the woodheating season and a -6% difference during the non-woodheating season. These results suggested greater losses of VOCs due to volatilization when woodburning emissions are more significant.

Concerns have been raised over the high sampling temperature used in the TEOM. Meyer et al. (1992) showed that the high sampling temperature may result in the partial volatilization of organic compounds such as those found in woodsmoke. A woodsmoke exposed quartz filter baked in a drying oven at 50°C showed a loss in mass of more than 25% after 4 hours of exposure, and 50% after 3 days. The blank control filter showed a loss of 14% after 3 days. Rupprecht & Patashnick (1993) suggested that lower sampling temperatures (e.g., 30°C) may be used to reduce these potential volatilization losses in situations where the contributions from volatile or semi-volatile components are expected to be significant. However, this is recommended only where outdoor temperatures do not exceed 25°C more than 5% of the time.

The effect of sampler temperature and flow rate was investigated by Meyer et al. (1995), in Salt Lake City, Utah during December 1994. A PM_{10} high-vol sampler was co-located with two TEOM monitors, one operating at 50°C and 3 L/min flow rate and the other at 30°C and 1 L/min flow rate. Face velocities of 41 cm/s were estimated for the hi-vol sampling, and 44 cm/s and 15 cm/s for the higher- and lower-volume TEOM instruments. In a comparison of the TEOM instrument operating at 30°C with that operating at 50°C, the authors reported a ratio of 2.23 (±0.22) for the month of December. This finding suggests that by lowering the equilibration temperature by 20°C and reducing the flow rate by a factor of 3, more than 2 times as much PM_{10} is retained on the filter. Comparisons of the two TEOM instruments with the hi-vol PM_{10} sampler produced ratios of 1.66 (±0.34) and 0.74 (±0.14), respectively, indicating a wide variation among the samplers used. More significantly, these findings suggest that the reference sampler does not measure all of the ambient PM_{10}.

3.2.5 Integrating Nephelometers

The integrating nephelometer is commonly used in visibility studies. It measures the optical scattering coefficient from the light scattered by particles and gases in the air, integrated over a wide range of angles. It is composed of an enclosed volume painted black, a light source on one side of the volume, and a light detector looking through the volume. The light received by the detector is that which has been scattered within the volume. Those particles in the size range of 0.3-0.7 µm are the most efficient at scattering visible light.

The range and sensitivity of integrating nephelometers are dependent on the individual instrument optics, electronic design and mode of operation. However, particle scattering coefficients can typically be measured over the range of approximately 1×10^{-6} to 1×10^{-2} m^{-1}, and commercial instruments typically have a sensitivity of about 0.5×10^{-6} m^{-1} (Lodge, 1989).

Larson et al. (1992), found that in woodsmoke-impacted areas, where the fine particle mode is expected to dominate, weekly averaged nephelometer measurements showed a high degree of correlation (r^2=0.945) with ambient $PM_{2.5}$ measurements. However, while relative loadings of $PM_{2.5}$ can be inferred from the backscatter measurements, this instrument is relatively insensitive to larger particles.

3.2.6 Single Particle Samplers

The measurement of particle size distributions is useful for studies involving particle sizing and collection transport and transformation, source characterization, and particle sizing and collection device performance (US EPA, 1996). Whereas cascade impactors can obtain particle distributions in terms of mass, single particle counters obtain distributions based on particle count. By making assumptions regarding particle shape and density, surface area and volume distributions may then be calculated.

The three most commonly used types of single particle samplers are aerodynamic particle sizers, electrical mobility analyser and optical particle counters (OPC's). Each is briefly described below.

Aerodynamic particle sizers use two parallel laser beams to measure the velocity lag of particles suspended in accelerating air flows (APS 3310, TSI. Inc., St. Paul, MN). Particle velocity is a function of particle diameter as long as the particle Reynolds number, Rep, which describes the gas flow around a particle, remains small (Wilson and Liu, 1980). As Rep increases, particle density and shape become additional factors.

This instrument generally allows a rapid and precise measurement of the aerodynamic size of most particles, and is typically applied in studies where particle diameters are larger than 0.5 µm. Inaccuracies may be encountered in measuring the aerodynamic diameter of non-spherical particles, due to the high acceleration in the instrument's acceleration nozzle (Baron et al., 1993). Coincidence errors, in which two particles are detected as a single particle, may be experienced at high particle concentrations (Heitbrink et al., 1991).

In electrical mobility analyzers, particles with a known charge distribution migrate through an electric field. Particle mobility is related to particle size. Electrical mobility analyzers are generally limited to studies where particle size is in the submicrometre range and particles are approximately spherical in shape. Examples of this technology are the electrical aerosol analyser (EAA) (Whitby and Clark, 1966) and the differential mobility analyzer (DMA) (Knutson and Whitby, 1975a, b).

The EAA uses unipolar diffusion charging and measures the integrated band of mobility. Standard data reduction tables are used to relate particle size, particle charge and electrical mobility under standard conditions. Yeh (1993) describes two major limitations with respect to EAAs. First, a minimum exists in the electrical mobility for given diffusion charging conditions as a function of particle size. This minimum lies between 1.0 and 2.0 µm particle diameter. To avoid errors in electrical mobility measurements due to larger particles, use of an aerosol preclassifier such as an impactor is recommended where particle diameters larger than 1.0 µm are expected. The second item of note is that because the reduction tables are valid only for standard operating conditions, any deviations from these conditions may lead to errors.

The DMA uses an equilibrium charge state of the particles, which is considered more stable than the distribution obtained from unipolar diffusion charging, and measures the extracted narrow band of mobility. As a result, the DMA has better resolution than the EAA. In addition, Yeh (1993) shows that the theoretical basis for predicting the equilibrium charge distribution is better understood and more accurate than that for diffusion charging theory. A limitation of the DMA is with respect to the neutralizer required to bring particle charge distribution to Boltzmann equilibrium. Radioactive sources such as a-emitters 241Am or 210Po and b-emitter 85Kr are commonly used as neutralizers. Yeh (1993) cites studies by Leong et al. (1983) and Winklmayr et al. (1990) as indicating that radiolytic processes can result in the formation of ultrafine particles, thereby arising as an artifact in size measurements.

OPCs measure the light scattered by individual particles traversing a light beam. The signals are processed in a multi-channel analyser, where light flashes are converted into electrical pulses. Number concentration is then derived from the count rate of the pulses and particle size is derived from the pulse height. OPCs are typically used to measure the number of particles in the size range of 0.1-50 μm. In the submicrometre range, counting efficiency decreases with decreasing particle size. In a review of OPC instruments, Gebhart (1993) reports that instruments using incandescent light count particles of 0.3-2 μm diameter with an efficiency of less than 20%. Better performance is observed with laser instruments, which typically exhibit a counting efficiency of 100% for particle diameters down to 0.1 μm. OPCs can experience coincidence errors and counter saturation at high particle concentrations.

3.3 PARTICLE SAMPLING SYSTEMS

To provide the type of detailed chemical analysis required for source apportionment studies, specialized particle sampling systems have been developed. They typically consist of multiple inlets, denuders and filters in parallel or series. A range of filter media is also typically used to optimize analyses for various chemicals. However, the care required to maintain these systems and the costs required to carry out the laboratory analyses limit their general application.

The University of California at Davis designed a specialized device for a cooperative visibility monitoring effort in Class I areas of the United States (Eldred et al., 1988). The study was known as the Interagency Monitoring of Protected Visual Environments (IMPROVE) and the samplers have since been called IMPROVE samplers. These units have been applied throughout North America, with intensive studies carried out in the Lower Fraser Valley (Sakiyama, 1994; Pryor and Barthelmie, 1996).

Many different sampling systems have been developed or modified for specific studies, including the Southern California Air Quality Study (SCAQS) (Fitz et al., 1989), and the Valley Air Quality Study (VAQS) (Chow et al., 1993b). Chow (1995) provides a list of several filter-based particle sampling systems and cites references to the associated research studies.

3.4 PERSONAL EXPOSURE MONITORS

Personal exposure monitors (PEMs) are sampling devices worn on the body to estimate an individual's exposure to air pollutants. As such, they provide a better representation of what individuals actually breathe than do fixed outdoor monitors or even indoor monitors. Due to the nature of their application, PEMs must meet a number of additional criteria relative to fixed-location samplers: low noise; light weight; portability; rugged design; ease of operation; adequate battery lifetime to meet sampling requirements; and comparability with fixed-location monitors. PEM design is typically based on cyclone or impactor samplers. Because of the lower flow rates and poorer flow controllers used in these devices, they are believed to have poorer precision than ambient samplers (Wiener and Rodes, 1993).

Lioy et al. (1990) employed a single-stage PM_{10} impactor called a lollipop during Phase II of the Total Human Environmental Exposure Study (THEES). The sampler was designed to collect particles on 25 mm filters at a flow rate of 4 L/min. During performance tests, five side-by-side personal impactors were operated close to three 10 L/min Indoor Air Sampling Impactors (IASI) (Marple et al., 1987) and one dichot sampler. Correlation coefficients near 1 were obtained, suggesting that within the test limits, the prototype samplers provided accurate estimates of PM_{10} concentrations. The Particle Total Exposure Assessment Methodology (PTEAM) study used a PM_{10} impactor designed by the University of Minnesota to operate with 37 mm filters and a flow rate of 4 L/min (Wiener et al., 1990). During the large-scale field study, PEM measurements taken from a central outdoor site averaged about 5% lower than 85 paired measurements made with co-located dichot samplers (Wallace et al., 1991).

Colome et al. (1992) used cyclone samplers (SKC Inc., Fullerton, CA) as PEMs in a study involving primarily asthmatics in Orange County, California. The cyclone had a 50% cut point of approximately 5 μm and excluded particles larger than 7 μm. A portable constant-flow battery-operated pump was used. The cyclone and pump assembly operated with 37 mm

Teflon filters at a flowrate of 2 L/min. In a comparison with PM_{10} measurements made concurrently with a Marple PM_{10} sampler (Marple et al., 1987), nearly 90% of the variation in PM_{10} mass was explained by the PEM measurements. Colome et al. (1992) concluded that for this application, the cyclone sampler provided a good and inexpensive measure of PM_{10} mass.

3.5 SITING CRITERIA

Monitoring data obtained from fixed samplers represent pollution levels at an individual site and not necessarily levels representative of an entire community. Each site is unique with respect to sensor location, surrounding structures and land use patterns, local meteorology, sources and other parameters that will affect measured pollution levels. Therefore, knowledge of site characteristics is important when comparing pollution levels among different sites.

Site documentation for the NAPS monitoring network has been compiled by Environment Canada (1988). A description of the site and nearby sources, a site map and photographs are provided for each station.

In the NAPS network, stations are sited based on population, a demonstrated need for monitoring or other special pollution problems of national importance, such as long-range pollutant transport (Environment Canada, 1988). Based on the station class and location, recommended criteria are provided for the spatial scale of representativeness. Additional requirements for probe siting are provided for the various criteria air pollutants. As such, requirements for particulate monitoring refer to TSP and not PM_{10}, but are applicable to the latter. Criteria include the following:

- probe height 2-15 m above ground,
- distance from supporting structure >2 m (horizontal),
- distance from trees >20 m,
- distance from sampler to obstacle such as buildings must be at least twice the height of the obstacle protruding above the sampler,
- unrestricted air flow in 3 out of 4 cardinal wind directions,
- no furnace or incineration flues should be nearby and
- spacing from roads varies with height of monitor.

More information on individual sampler locations is provided in Chapter 5 (Table 5.3).

3.6 SUMMARY

Reflecting the wide spectrum of physical and chemical characteristics associated with particulate matter, several techniques are available to determine directly or indirectly mass concentrations of particulate matter. Therefore, it is important to understand the capabilities and limitations of the various methods, and to be able to relate the measurements made by one method to another.

Methods that are currently used to directly measure the mass concentration of PM_{10} in the atmosphere include SSI samplers, dichot samplers and Partisol samplers. $PM_{2.5}$ measurements are available through dichot and Partisol samplers and also various cyclone samplers and virtual impactors developed to monitor smaller size fractions. In each case, air is drawn through a filter over a known period. Further speciation of the collected material is possible through analysis of the soiled filter. Samplers may vary as to inlet design, operating flow rates and collection media. Intercomparison studies of PM_{10} samplers show that inter-sampler differences are minimized to within predicted levels when the samplers are regularly maintained. Good agreement has been observed between dichot, Partisol and Xontech/AirMetrics samplers for $PM_{2.5}$ measurements.

Indirect methods measure parameters other than mass that can be later converted to obtain units of mass concentration. The BS sampler and the tape sampler employ somewhat similar methodologies. In both cases a filter or tape is exposed to the atmosphere for a set time, after which the reflectance or transmittance of light through the soiled substrate is measured and related back to mass. Both methods are most sensitive to the light-absorbing fractions of particulate matter in the range of 0 to about ≤ 3.5 - 4.5 µm in diameter and, as such, are poor representatives of PM_{10}, except where this component dominates.

Automated indirect methods approved by the US EPA include the beta-attenuation (or beta-gauge) monitor and the TEOM monitor. In the beta-attenuation monitor, mass determination is based on the attenuation that a beta-ray particle undergoes as it passes through an exposed filter. In the TEOM, air is drawn through a filter and down a tapered tube, which will oscillate at a frequency that can be related to mass collected on the filter. Both can provide almost real-time measurements. Both have shown a tendency to underestimate concentrations obtained from manual samplers. There are some concerns that the high TEOM sampling temperature (30°C or 50°C) can result in the loss of semi-volatile species such as nitrates and various organic compounds, as observed in woodsmoke-impacted areas during the wintertime. These underestimates are particularly significant when sampling for $PM_{2.5}$. The integrating nephelometer measures the scattering of light by particles, and can provide a relative measure of $PM_{2.5}$ loadings in areas where fine particles dominate.

Sampling systems developed for the IMPROVE, VAQS and SCAQS programs are examples of systems that use multiple inlets, denuders and filters to enable detailed chemical analyses to be carried out. Although PEMs are worn on the body and are therefore more representative of what an individual breathes, they represent unique challenges in that they require compact, lightweight and portable design that gives results comparable to ambient samplers.

4 SOURCES OF PM

PM may be discharged directly to the atmosphere from a broad range of sources, both natural and anthropogenic, or it may be produced in the atmosphere via secondary reactions. As discussed in Chapter 2, the size and chemical composition of the particles formed will reflect the parent materials and the formation processes involved. High-temperature combustion processes and secondary reactions typically produce particles in the fine fraction ($D_p \leq 2.5$ µm), while natural and anthropogenic sources involving mechanical processes contribute more greatly to the coarse fraction ($D_p > 2.5$ µm).

Table 4.1 (from Chow, 1995) identifies the dominant size fraction and chemical species associated with various source types. There is a clear distinction in both particle size and chemical composition between those particles derived from fugitive dust sources, and those resulting from high-temperature combustion processes. Fugitive dust sources characteristically emit coarse particles that are predominantly composed of crustal elements such as Si, Al, K, Ca and Fe. In contrast, combustion and metal refining processes emit fine particles that are high in organic and elemental carbon, SO_4^{2-} NH_4^+ and NO_3^-.

In the following, the major sources of PM_{10} are discussed. Sources contributing more significantly to the fine fraction are described in Section 4.1, and those contributing more to the coarse fraction are described in Section 4.2. Precursors to secondary particles are discussed in Section 4.3. Provincial and national emission estimates are presented in Section 4.4, and associated uncertainties are described in Section 4.5. A summary is provided in Section 4.6.

4.1 PRIMARY SOURCES OF THE FINE FRACTION

4.1.1 Prescribed Burning and Wildfires

Prescribed burning is used as a tool for hazard reduction, site preparation, wildlife habitat improvement, disease and insect control and ecosystem maintenance (Smith and Stoneman, 1992). Particle emission rates vary depending on fuel type and on the phase of combustion (flaming, smoldering, or a combination of the two). Total particle emissions have been found to increase by a factor of 2 - 4 in going from flaming to smoldering (Susott et al., 1991). Prescribed burning and wildfires are a major source of fine particles in western North America (Lamn et al., 1992).

Generally, 90% of particles from prescribed fires are less than 10 µm in diameter, and approximately 70% are less than 2.5 µm in diameter (Lamn et al., 1992). However, the actual size distribution of particles will vary depending on the rate of energy release of the fire. In lower-intensity fires, a normal distribution with a peak near 0.3 µm is observed (Ward and Hardy, 1984). In high-intensity fires, a bimodal distribution is observed, characterized by peaks near 0.03 µm and 35+ µm. The larger particles reflect the increased mechanical turbulence present in such fires.

The fine fraction consists of approximately 40-70% organic carbon material, 2-15% graphitic carbon, and the remainder inorganic ash material (Ward and Blakely, 1992). A variety of PAHs have been identified in the organic fraction, including anthracene, phenanthracene, benz[a]anthracene, chrysene, fluoranthene, and benzo[a]pyrene. Potassium is the most abundant trace metal in the plumes. Measurements show that K, S and Cl are highest during flaming stages, and Al, Si, Ca and Fe tend to increase during the smoldering phase (Susott et al., 1991).

4.1.2 Residential Wood Combustion

Residential wood combustion (RWC) is a major source of wintertime particle emissions in communities where there is a high density of woodstove users and they are subject to persistent temperature inversions and therefore poor air dispersion. Several studies on RWC have been conducted in the United States: Puget Sound, Washington (Maykut and Fry, 1992), Mammoth Lakes, California (Ono and Taylor, 1992), Medford, Oregon (Hough et al., 1992), Libby, Montana (Morris et al., 1992; Manderino et al., 1992), Denver, Colorado (Schwayder, 1992), and Waterbury, Vermont (Sexton et al., 1985). Similar studies in Canada are limited. Crozier and Manna (1988)

Table 4.1 Characterization of PM_{10} from Various Sources (based on Chow, 1995)

Source Type	Dominant Particle Size	Chemical Abundances in Percent Mass			
		< 0.1%	0.1 to 1%	1 to 10%	> 10%
Motor Vehicle	Fine (0 to 2.5 μm)	Cr, Ni, Y, Sr, Ba	Si, Cl, Al, Si, P, Ca, Mn, Fe, Zn, Br, Pb	Cl^-, NO_3^-, SO_4^{2-}, NH_4^+, S	Organic Carbon (OC), EC
Vegetative Burning	Fine	Ca. Mn, Fe, Zn, Br, Rb, Pb	NO_3^-, SO_4^{2-}, NH_4^+, Na^+, S	Cl^-, K^+, Cl, K	OC, EC
Residual Oil Combustion	Fine	K^+, OC, Cl, Ti, Cr, Co, Ga, Se	NH_4^+, Na^+, Zn, Fe, Si	V, OC, EC, Ni	S, SO_4^{2-}
Incinerator	Fine	V, Mn, Cu, Ag, Sn	K^+, Al, Ti, Zn, Hg	NO_3^-, Na^+, EC, Si, S, Ca, Fe	Si
Coal-Fired Boiler	Fine	Cl, Cr, Mn, Ga, AS, Se, Br, Rb, Zr	NH_4^+, P, K, Ti, V, Ni, Zn, Sr, Ba, Pb	SO_4^{2-}, OC, Ec, Al, S, Ca, Pb	Si
Oil-Fired Power Plant	Fine	V, Ni, Se, AS, Br, BA	Al, Si, P, K, Zn	NH_4^+, OC, EC, Na, Ca, Pb	S, SO_4^{2-}
Smelter Fine	Fine	V, Mn, Sb, Cr, Ti	Cd, Zn, Mg, Na, Ca, K, Se	Fe, Cu, AS, Pb	S
Antimony Roaster	Fine	V, Cl, Ni, Mn	SO_4^{2-}, Sb, Pb	S	None reported
Paved Road Dust	Coarse (2.5 to 10 μm)	Cr, Sr, Pb, Zr	SO_4^{2-}, Na^+, K^+, P, S, Cl, Mn, Zn, Ba, Ti	Elemental Carbon (EC), Al, K, Ca, Fe	(OC), Si
Unpaved Road Dust	Coarse	NO_3^-, NH_4^+, P, Zn, Sr, Ba	SO_4^{2-}, Na^+, K^+, P, S, Cl, Mn, Ba, Ti	OC, Al, K, Ca, Fe	Si
Construction	Coarse	Cr, Mn, Zn, Sr, Ba	SO_4^{2-}, K^+, S, Ti	OC, Al, K, Ca, Fe	Si
Agriculture Soil	Coarse	NO_3^-, NH_4^+, Cr, Zn, Sr	SO_4^{2-}, Na^+, K^+, S, Cl, Mn, Ba, Ti	OC, Al, Mg, K, Ca, Fe	Si
Natural Soil	Coarse	Cr, Mn, Sr, Zn, Ba	Cl^-, Na^+, EC, P, S, Cl, Ti	OC, Al, Mg, K, Ca, Fe	Si
Lake Bed	Coarse	Mn, Sr, Ba	K^+, Ti	SO_4^{2-}, Na^+, OC, Al, S, Cl, K, Ca, Fe	Si
Marine	Fine and coarse	Ti, V, Ni, Sr, Zr, Pd, Ag, Sn, Sb, Pb	Al, Si, K, Ca FE, Cu, Zn, Ba, La	NO_3^-, SO_4^{2-}, OC, EC	Cl^-, Na^+, Cl, Na

investigated the effects of RWC on air quality in Cranbrook, British Columbia.

Approximately 95% of particles emitted from woodstoves are reported to be less than 0.4 μm in diameter and therefore almost entirely in the fine fraction (Rau and Huntzicker, 1984).The MMAD of particles generated from wood-burning fireplaces is approximately 0.17 μm (Dasch, 1982).

Woodstove emissions are predominantly consisting of organic carbon, elemental carbon and sulphate, with trace amounts of potassium, chloride and nitrate. More than 100 different organic compounds have been identified in woodsmoke. A major constituent of the PM_{10} fraction is polycyclic organic material (POM) which results from the incomplete combustion of hydrocarbons. This group includes PAHs, polycyclic heterocyclic compounds and various derivatives. PAH emission rates from woodstoves are substantially higher than from prescribed burning and fireplaces. Softwood combustion has been found to emit more elemental carbon than hardwood combustion which is primarily composed of organic compounds (Dasch, 1982).

Elemental potassium-to-iron ratios ranging from 15-230 have been found in woodsmoke, while most other sources exhibit ratios of <0.35 (Wolff et al., 1981;Watson, 1979).This observation is used to trace particle emissions to woodsmoke sources.

4.1.3 Transportation Sources

The transportation sector has been identified as a large source of PM, generating emissions through fuel combustion and engine wear. Motor vehicles also generate PM through tire and brake wear and the resuspension of road dust.

Particle emissions from on-road motor vehicles are typically subdivided into the following types: exhaust, tire wear, brake lining and road dust. Both gasoline and diesel vehicles produce particle emissions that are almost entirely <10 μm and predominantly in the fine fraction. In studies carried out in the United States, an estimated 89% and 92% of particle emissions from light-duty gasoline and diesel vehicles were smaller than 2.5 μm (Cooper et al., 1987). Comparable studies in Canada are lacking. As Canadian vehicles differ as to onboard emission controls and fuel characteristics, emission characteristics may differ from their US counterparts.

Measurements show that mass emission rates from diesel trucks are 6-100 times higher than those from gas automobiles (Hildemann et al., 1991a; NRCC, 1982; Williams et al., 1989), while noncatalyst cars emit more submicrometer particles than catalyst cars (Hildemann et al., 1991a). The size distribution for the submicrometre fraction of automobile emissions is unimodal and peaks at about 0.1 μm, although bimodal distributions are also observed (Hildemann et al., 1991a). Diesel truck emissions also exhibit a unimodal size distribution in the submicrometer range, peaking around 0.2 μm.

Chemical analyses of fine (<2 μm) particles in exhaust from noncatalyst and catalyst-equipped automobiles and heavy-duty diesel trucks currently in use in the Los Angeles area have identified more than 100 different organic compounds. These compounds include n-alkanes, n-alkanoic acids, benzoic acids, benzaldehydes, PAH, oxy-PAH, steranes and pentacyclic triterpanes (Rogge et al., 1993a). Chemical composition typically shows wide vehicle-to-vehicle variation. The organic carbon component has been estimated to account for 38-75% of the fine particle mass, while the elemental carbon component accounts for 4-37% of the total (Hildemann et al., 1991b). PAH and oxy-PAH emissions are highest from noncatalyst-equipped automobiles, while n-alkanoic acid emissions are higher from automobiles equipped with catalytic converters (Rogge et al., 1993a). Heavy duty diesel trucks emit four times as much PAH as catalyst-equipped vehicles, but nearly seven times less PAH than noncatalyst-equipped vehicles. Heavy-duty diesel trucks also emit substantial quantities of n-alkanes and n-alkanoic acids. Bromine is found in trace amounts (3%) in non-catalyst vehicle exhaust (Hildemann et al., 1991b). Particle sulphate is also found in trace amounts (4%) in vehicle exhaust. Diesel engines, which use a large amount of excess oxygen, have a greater tendency to form sulphates than gasoline engines (ASEP, 1997). However, the larger contribution is through the conversion of SO_2 emissions from vehicular exhaust to secondary particles. Reflecting the higher fuel sulphur content found in diesel fuel compared with gasoline (i.e., up to a factor of four difference), higher SO_2 emissions are observed from the diesel vehicle fleet as compared with the gasoline vehicle fleet (e.g., MELP, 1995; ASEP, 1997). Vehicular exhaust is also a large source of NO_x emissions that may convert in the atmosphere to particle nitrate.

Additional primary mobile particle emissions are attributed to tire wear, brake linings and re-suspended fugitive dust. Analyses have shown that brake dust consists primarily of iron and organic carbon compounds (11% each), magnesium (8%), barium and silicon (7% each), sulphates (3%) and elemental carbon (3%) (Hildemann et al., 1991b). Similar analyses of tire dust show that organic carbon (15%) and elemental carbon (36%) are the dominant constituents. However, a large fraction of the total mass is not accounted for in these estimates.

Particulate lead (from the octane enhancer tetraethyl lead) and bromine (from the lead scavenger ethylene dibromide) have traditionally been used to identify particle emissions from automobiles. However, leaded gasoline was phased out in Canada and in many parts of the United States in 1990. With the phase-out of leaded gasoline, new tracer species have become necessary. In Canada, methylcyclopentadienyl manganese tricarbonyl (MMT) has been used as an octane enhancer in gasoline since 1977. Combustion results in the formation of various oxides of manganese. In a study carried out in Montreal, Loranger and Zayed (1992) found that ambient manganese levels appeared to correlate well with traffic density in an urban setting. However, the difficulty in using manganese as a tracer for gasoline vehicles is that other sources of manganese exist in the environment, such as crustal material. Steranes and pentacyclic triterpanes, which have been extensively used in geochemistry as petroleum markers, are introduced into vehicular emissions via engine oil. These compounds have been identified as potential markers for internal combustion engines in Southern California (Rogge et al., 1993a). Different PAH signatures have been used to differentiate among emissions from spark ignition engines, diesel engines and residential wood combustion (Li and Kamens, 1993). In an airshed where emissions are predominantly from motor vehicles and residential wood combustion, several VOCs were identified as potential tracers for motor vehicle emissions, including xylenes; methylcyclohexane; 2- and 3-methylhexane; 2,3,4- and 2,2,4-trimethylpentane; and 2-methylpentane (Zweidinger et al., 1990).

4.1.4 Fossil Fuel Combustion

Fuel combustion typically includes all boilers, heaters and furnaces associated with utilities, industry, and commercial/institutional and residential establishments. It also includes residential wood combustion. Emissions from fuel combustion processes are dependent on many factors, including the type of fuel burned, the source type and the type of emission controls employed.

Coal is a slow-burning fossil fuel characterised by a high ash content. The combustion of coal is a large source of PM in the utility and industrial sectors. Emissions depend in part on the type of coal burned and its ash content. For instance, the combustion of low-ash coal yields less PM than that of high-ash coal. Major chemical constituents of PM from coal combustion include oxides of silicon, aluminum and iron, with lesser amounts of sulphur, magnesium, potassium and calcium. Particles <1 µm in diameter are enriched in volatile elements such as arsenic, antimony, selenium and gallium, indicative of material vaporized during combustion (Markowski and Filby, 1985). The presence of selenium is of particular note, as coal combustion is estimated to be the largest source of atmospheric elemental selenium in the United States, with contributions of 60-85% of anthropogenic emissions (NAS, 1976; Eimutis et al., 1978). As the selenium content of coal is an order of magnitude greater than that of oil, this observation is used to discriminate between air masses originating over coal-burning areas in the Midwestern United States, and those originating over oil-burning regions of the east coast (Spengler and Thurston, 1983). During the summer of 1986 in southern Ontario, a study of transported acid PM identified coal combustion as the largest single contributor (75%) to the fine particle mass (Keeler et al., 1990).

The predominant types of fuel oil burned by combustion sources are distillate oils and residual oils. Distillate oils, including Nos. 1 and 2 fuel oil, are more volatile and less viscous than residual oils. They contain little nitrogen or ash and less than 0.3% sulphur by weight. They are used mainly in domestic and small commercial applications. Residual oils, including No. 5 and No. 6 (Bunker C) fuel oil, contain significant quantities of ash, nitrogen and sulphur. The sulphur content of high-sulphur No.6 fuel oil is 3.97% by weight. They are used in utility, industry and large commercial applications. Measurements of emissions from an industrial-scale boiler burning No. 2 fuel oil showed a bimodal mass distribution, with peaks near 0.05 µm and 0.5 µm (Hildemann et al., 1991a). The mass distribution of particles resulting from heavy oil combustion was found to be shifted toward larger particle sizes, with peaks near 0.18 µm and 5.7 µm (Buerki et al., 1989). Analysis of the larger-diameter mode revealed spherical particles containing many holes, which is typical of fly ash.

Particle emissions from distillate oil fired boilers are typically carbonaceous in nature and dependent on the completeness of combustion. Emissions from residual oil combustion are high in carbon and sulphur levels and depend on oil sulphur content. Low-sulphur residual oils have a lower viscosity and reduced asphaltene, ash and sulphur content, resulting in better atomisation and fuel combustion. Usually, the combustion of distillate oils produces less PM than that of heavier residual oils. Reduced boiler loads may also contribute to decreased particle emissions.

Trace elements associated with oil combustion include cadmium, cobalt, copper, nickel, vanadium, sodium and iron. Vanadium is considered the key indicating species of residual oil combustion. In the United States, an estimated 90% of vanadium emissions are attributed to the combustion of residual oil (NAS, 1974). Although coal also contains some vanadium, its levels are much less than those found in residual oil.

4.1.5 Nonferrous Metal Industries

Nonferrous industries include the smelting of metals other than iron, and include the production of copper/nickel, lead/zinc and aluminum. PM from metal smelters is typically enriched in the so-called chalcophilic elements (e.g., sulphur, copper, zinc, arsenic, selenium, silver, cadmium, indium and gold) compared with the feed material. Emissions from copper smelters in particular have been found enriched in copper, arsenic, selenium, cadmium and indium. These characteristic chemical profiles have been used to identify the plumes and PM from individual smelters (Small et al., 1981).

Aluminum smelters are sources of various particle fluorides (cryolite, aluminum fluoride, calcium fluoride, chiolite), alumina and ferric oxide. They are also a source of vaporized organic compounds and sulphur dioxide, which later may be involved in secondary particle production. Particle emissions from lead smelters are composed of a range of materials, including lead oxides, quartz, limestone, iron pyrites, iron-lime silicate slag, arsenic and other metallic compounds. Much of the PM from zinc smelters is from the zinc roasters, in which zinc sulphide concentrate is converted to an impure zinc oxide called calcine. Emissions from such operations as crushing, grinding and material transport may also contribute fugitive emissions.

4.1.6 Pulp and Paper Industry

In chemical wood pulping, cellulose is extracted from wood by dissolving the lignin that binds the cellulose fibres together. Kraft, sulphite, neutral sulphite semi-chemical (NSSC) and soda (US EPA, 1992) are four major processes for chemical wood pulping. Particle emissions from Kraft pulp mills are primarily from the recovery furnace, the lime kiln and the smelt dissolving tank. They consist mainly of sodium salts and some calcium salts from the lime kiln, and are caused by carryover of solids and sublimation and condensation of the inorganic chemicals. Except for uncontrolled emissions from the lime kiln, particle diameters are predominantly less than 2.5 µm. In sulphite mills, a major source of PM is the absorption system used to recover SO_2 in the recovery furnace exhaust. Auxiliary power boilers fuelled by coal, oil or bark/wood residues may also be large sources of particle emissions in pulp mills.

4.1.7 Wood Products Industry

This industry involves the processing of wood-derived products. It includes sawmills and plywood and veneer manufacture. Plywood manufacturers are a source of both PM and organic matter. The major source is the veneer dryer, which emits various condensable and volatile organic compounds from the wood (US EPA, 1992). The condensable organic fraction consists largely of wood resins, resin acids and wood sugars that cool outside the stack to temperatures below 21°C and combine with water vapour to form a blue haze, a water plume or both. The volatile fraction consists of terpenes and, for gas-fired dryers, natural gas components. Plywood manufacturers may also be a source of fugitive emissions as sawdust and other small wood particles, primarily from plywood cutting and sanding operations.

4.2 PRIMARY SOURCES OF THE COARSE FRACTION

4.2.1 Windblown Dust

The action of the wind on soils and crustal materials results in the gradual erosion of the parent material, producing particles that become suspended in the atmosphere. The size distribution is typically bimodal in nature, with maxima reported at 1-10 µm and 50 µm (Gillette, 1980). More than 90% of the mass is greater than 1 µm in diameter (Gillette, 1980; Houck et al., 1989).

In a review of mass balance studies in the United States, Chow (1995) found that approximately one-half of PM_{10} consists of geological material, but only about 10% is $PM_{2.5}$. Reflecting the composition of the earth's crust, soil-derived material is typically composed of seven major elements: silicon, aluminum, iron, sodium, potassium, calcium and magnesium. Actual composition will vary from site to site; however, silicon, aluminum, iron and calcium are typically used as key indicators of soil-derived material (e.g., Lowenthal et al., 1994). Because the atmospheric concentrations of many trace elements tend to be a factor of 10-1000 times higher than expected from the physical dispersion of crustal material, enrichment factors are sometimes used to help identify the source of the PM. Enrichment factors are typically defined as the ratio of the airborne concentrations of element *x* to aluminum, divided by that in crustal rock. Ratios near unity suggest that crustal weathering is the source of element *x*, while ratios much larger than unity indicate noncrustal sources (Milford and Davidson, 1985).

4.2.2 Marine

Size distributions of marine particles are characterized by two distinct modes separated by a minimum in the range of 0.2-0.3 μm radius. The fine mode (<0.2 μm radius) comprises 90-95% of the particles but only about 5% of the particle mass. It consists primarily of non-sea salt sulphate (Fitzgerald, 1991). The coarse mode (>0.5 μm radius) comprises 90-95% of the total mass, but little of the total number of particles. This fraction is composed primarily of sea salt particles, which are generated at the surface of the ocean by the bursting of whitecap bubbles. Each bursting bubble produces about 1-10 jet drops and hundreds of film drops. Most jet drops have a radius of less than 10 μm, while most film drops have a radius smaller than 1 μm (Woolf et al., 1988). Depending on the relative humidity, the drops become either sea-salt particles or sea-salt solution droplets. Larger particles (>10 μm radius) known as spume drops are produced from the mechanical disruption of wave crests by the wind. Spume droplet formation becomes significant at wind speeds greater than 10 m/s (Wang and Street, 1978; Monahan et al., 1986). Sea salt particles are primarily composed of water and dissolved and suspended materials such as sodium chloride, potassium, calcium, organic compounds, sulphates and carbonates.

4.2.3 Vegetation

Vegetation-derived particles may be primary or secondary in nature. Seeds, pollen, spores, leaf waxes and resins are examples of particles emitted directly to the atmosphere. Particle size typically ranges from 1 to 250 μm (Warneck, 1988), with spore size between 5-10 μm (Corn, 1976b). Secondary particles are formed via reactions involving natural VOC's such as alpha- and beta-pinene released from vegetation (e.g., Hatakeyama et al., 1989; Zhang et al., 1992, Pandis et al., 1992). Further discussion of secondary particles is found in Section 4.3.

4.2.4 Road Dust

Road dust represents several source contributions ranging from vehicle exhaust, worn tires and brake linings particles, dirt from construction sites, blowing garden soil, leaves and other plant materials.

Sanding material has been shown to contribute almost half the PM_{10} emissions during the late winter/early spring snow melt in Telluride, Colorado (Dresser and Baird, 1988). Fine particle (<2 μm) paved road dust has been identified as the third largest source of fine organic carbon particle emissions in the Los Angeles Basin (Hildemann et al., 1994). Studies in the Denver metropolitan area show that PM_{10} emissions from clean, and paved roads account for about 40-70% of total PM_{10} (Zimmer et al., 1992).

Chemical analyses of paved road dust in the Los Angeles Basin show that the major chemical constituents include organic carbon compounds (13%), silicon (12%), iron (6%), aluminum (6%), calcium (4%), potassium (2%) and sulphate and elemental carbon (1% each) (Rogge et al., 1993b). Vehicular emissions, tire dust and vegetative detritus respectively contribute 7.6%, 1.6% and a minimum of 2.2% of the total fine road dust, and 32.7%, 4.3% and 5.3% of the organic component. Relative to ambient PM, road dust deposits are more typically enriched in mechanically generated fine particles such as leaf dust and soil dust, which deposit more readily by sedimentation or impaction. They contain hundreds of organic compounds, including n-alkanes (C_{19}-C_{39}), n-alkanoic acids (C_6-C_{32}), n-alkanal (C_{28}, C_{30}, C_{32}), n-alkanols (C_{26}, C_{28}), benzoic acid and its alkyl-substituted homologues, PAH and oxy-PAH, steranes, triterpanes, pesticide and insecticide residues and monoterpenoids.

4.2.5 Agriculture

Agricultural crops (wind erosion and tillage), dairy operations, feedlots and agricultural burning are potential sources of PM. Particle emissions from dairies and feedlots are typically larger than 2.5 μm and contain higher levels of organic carbon, nitrate and ammonium than agricultural soils (Houck et al., 1989). These chemical constituents are concentrated in the finer size fractions, indicating the importance of secondary organic and ammonium nitrate formation resulting from agricultural emissions.

Agricultural burning is performed for waste reduction, sanitation and maintenance purposes (Freeburn and

Schmitt, 1992). In contrast to other agricultural operations, agricultural burning (wheat and barley stubble) has been found to produce fine particle emissions, with an estimated 82% smaller than 2.5 µm and 93% smaller than 10 µm (Houck et al., 1989).These emissions are high in organic carbon, elemental carbon, chlorine and potassium.

4.3 SECONDARY PARTICLES

Secondary particle formation is dependent on many chemical reactions and transformations taking place in the atmosphere. The principal precursor gases involved in secondary particle formation include SO_2, NO_x, organic compounds and ammonia, which undergo reactions to yield such products as NH_4HSO_4, $(NH_4)_2SO_4$, various condensible organic compounds and NH_4NO_3.

The mechanisms for particle formation involving SO_2 and NO_x were described in Chapter 2. Briefly, sulphate is formed through the oxidation of SO_2 in the gas phase or aqueous phase. Gas-phase oxidation is primarily via reaction with the hydroxyl radical to form sulphuric acid gas, which may nucleate in the presence of water vapour to form sulphuric acid droplets. Sulphuric acid gas may also condense on existing particles. Important sources of SO_2 include the nonferrous mining and smelting sector and electric power generation. Particle nitrate is formed from reactions involving HNO_3 and either ammonia or pre-existing particles. Nitric acid is formed primarily from gas-phase reactions with the hydroxyl radical. Motor vehicles and electric power generation are large sources of NO_x. Estimates of both SO_2 and NO_x emissions are provided in Section 4.4.

Mechanisms for organic particle formation are less well understood. The potential for a particular VOC species to be involved in secondary particle formation will depend on the ease with which it can be oxidized and the volatility of its byproducts. Grosjean and Seinfeld (1989) used the results of smog chamber experiments to estimate the fraction of VOC converted into particles. This dimensionless number is called a fractional aerosol coefficient (FAC). Based on the compiled FACs, the most potent particle precursors were aromatic hydrocarbons, higher-molecular-weight olefins and cyclic olefins, and higher-molecular-weight paraffins including cycloalkanes. Applying this methodology to VOC emission estimates for a smog episode in Southern California, Grosjean (1992) determined that aromatics such as toluene, xylene and various substituted benzenes, and various VOCs associated with biogenic emissions yields the greatest quantity of secondary organic PM. In contrast, alkanes with up to six carbon atoms, all alkenes with up to six carbon atoms, benzene and many low-molecular-weight carbonyls, chlorinated compounds and oxygenated solvents do not form particles under atmospheric conditions.

Biogenic sources have long been associated with the blue haze observed in forested areas (Went, 1960). The study of the degradation pathways of biogenic hydrocarbons is an important and current problem in atmospheric chemistry (Zhang et al., 1992). Major VOC types emitted by biogenic sources include isoprene from deciduous trees and the monoterpenes alpha- and beta-pinene from coniferous trees (Lamb et al., 1986). Smog chamber studies have shown that the oxidation of isoprene contributes little to particle formation (Pandis et al., 1991). This finding is attributed to the high vapour pressure of the products of formation. In similar studies involving alpha-pinene (Hatakeyama et al., 1989; Zhang et al., 1992) and beta-pinene (Pandis et al., 1992; Hatakeyama et al., 1989; Zhang et al., 1992), measurable particle yields were detected. It was concluded that alpha- and beta-pinene could contribute to particle formation in areas with high vegetative coverage (Pandis et al., 1992).

Ammonia is the most common alkaline component in the atmosphere. It neutralizes such atmospheric acids as HNO_3, H_2SO_4 and hydrochloric acid (HCl) to yield several salts, including NH_4NO_3, $NH_4\,HSO_4$, $(NH_4)_2\,SO_4$ and ammonium chloride (NH_4Cl). For acid rain falling over large parts of Europe, up to 70% of the original acid is neutralized by ammonia (EMEP/CCC, 1984). Ammonia is emitted from many sources, including combustion processes, refinery operations, chemical fertilizer manufacture and application, chemical plants, steel mill coke ovens and decomposing livestock wastes. Livestock operations are typically the major source of ammonia emissions, contributing more than 50% of ammonia emissions in the South Coast Air Basin (Russell et al., 1983) and more than 80% in Europe (Buijsman et al., 1987). Similar estimates for Canadian sources are not available.

4.4 EMISSION ESTIMATES

Emission inventories are typically prepared using several methodologies, including:

- actual source measurements,
- emission factors applied to surrogate activities,
- raw data from industry and government agencies, and
- engineering estimates based on a fundamental understanding of the emission source.

The availability of actual source measurements is typically limited to large industrial facilities, due to the large costs associated with sampling programs. Particle emission estimates are obtained through noncontinuous sampling studies, and so provide only a snapshot of emissions for a particular set of plant operating conditions. Consequently, average emission factors are more commonly applied. Emissions may be calculated using the following formula:

$$E = \sum_i A_i \cdot F_i \cdot (1 - C_{eff,i})$$

where E is the emission estimate, A_i is the activity rate for source i, F_i is the emission factor for source i and $C_{eff,i}$ is the efficiency of control measures applied to a source i. For a point source, a typical activity rate would be per volume of fuel used, while a corresponding emission factor would be tonnes of pollutant per volume of fuel used.

In the 1990 national emission inventory for criteria contaminants (which included TSP but not PM_{10}), standard and nonstandard emission factors were used for 46.0 and 41.7%, respectively, of the calculations (Deslauriers, 1996b). Provincial permit information was used for 7.4% of the calculations. Emissions based on source testing or other measurements, material balance using engineering expertise and knowledge, specific reports and other methods accounted for less than 2% each of the total calculations.

Standard emission factors were derived from the 5th edition of the US EPA publication AP-42 (US EPA, 1996). For on-road vehicles, emission factors for gaseous pollutants (in units of gm/mi) were calculated from the Canadian version of the US EPA Mobile5 model, designated M5C. Corresponding TSP emission factors were based on standard factors in AP-42.

Biogenic emissions were estimated using CANBEIS (Canadian Biogenic Emission Inventory System), a modeling system based on the US EPA's system PC-BEIS (Personal Computer Biogenic Emission Inventory System). CANBEIS integrates the programs developed to estimate emission factors with a Geographical Information System (GIS) called SPANS, which includes land use and land cover information.

To estimate contributions from PM_{10} and smaller size fractions, additional speciation factors were applied to TSP emission estimates. These speciation factors were largely based on those included in the US EPA SPECIATE database (Radian Corp., 1993).

Estimates of primary particle emissions in Canada for the year 1990 have been prepared for PM, PM_{10}, $PM_{2.5}$ and $PM_{2.5\text{-}10}$ (Deslauriers, 1996b). Contributions from the major source categories are listed in Table 4.2. Sources considered in each category include the following:

- industrial: coal industry, pulp and paper industry, nonferrous mining and smelting, iron and steel production, wood industry and mining and rock quarrying,
- Nonindustrial fuel combustion: commercial and residential fuel combustion, residential fuel wood combustion and electric power generation,
- transportation: gasoline and diesel vehicles and trucks, propane-powered vehicles, railroads, marine craft, aircraft and tire wear,
- incineration: wood waste and other types of incineration,
- biogenic: forest fires,
- miscellaneous sources: structural fires, pesticides and fertilizer application, cigarette smoking and the marine cargo handling industry.

Emission estimates for open sources such as paved and unpaved roads, construction sites, agricultural and prescribed burning are currently under revision, and so excluded from Table 4.2. In 1990, an estimated 1.0 Mt of PM_{10} was emitted from primary sources excluding open sources. $PM_{2.5}$ accounted for approximately 75% of this total. Emission estimates for PM_{10} and $PM_{2.5}$ are further summarized in Figure 4.1. The source sector breakdown for PM_{10} emissions is as follows: 42% industrial sources, 28% forest fires, 15% nonindustrial fuel combustion, 11% transportation and 2% each incineration and miscellaneous sources. The corresponding breakdown for $PM_{2.5}$ emissions shows equally large (34%) contributions from forest fires and industrial sources, and

Table 4.2 1990 Emissions of PM in Canada (Deslauriers, 1996a)

Category/Sector*	PM tonnes	$PM_{2.5}$ tonnes	$PM_{2.5\text{-}10}$ tonnes	PM_{10} tonnes
Industrial Sector	810,366	270,182	178,509	448,691
Nonindustrial Fuel Combustion	272,842	126,513	31,041	157,554
Transportation	133,489	101,493	13,896	115,389
Incineration	34,248	13,683	5,128	18,811
Forest Fires	293,123	263,811	26,381	290,192
Miscellaneous	30,430	11,026	7,105	18,131
TOTAL	**1,574,498**	**786,708**	**262,060**	**1,048,768**

*Open sources and secondary particles are not included.

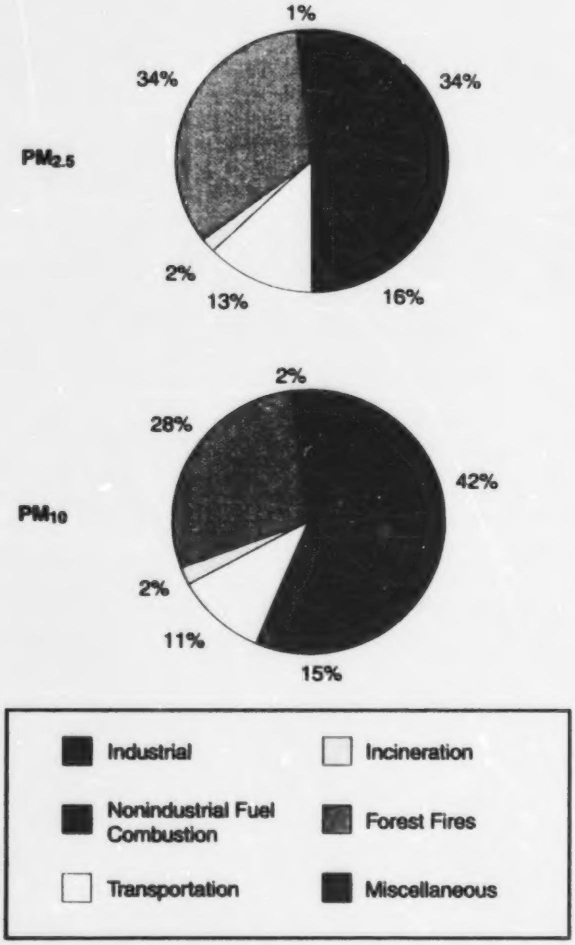


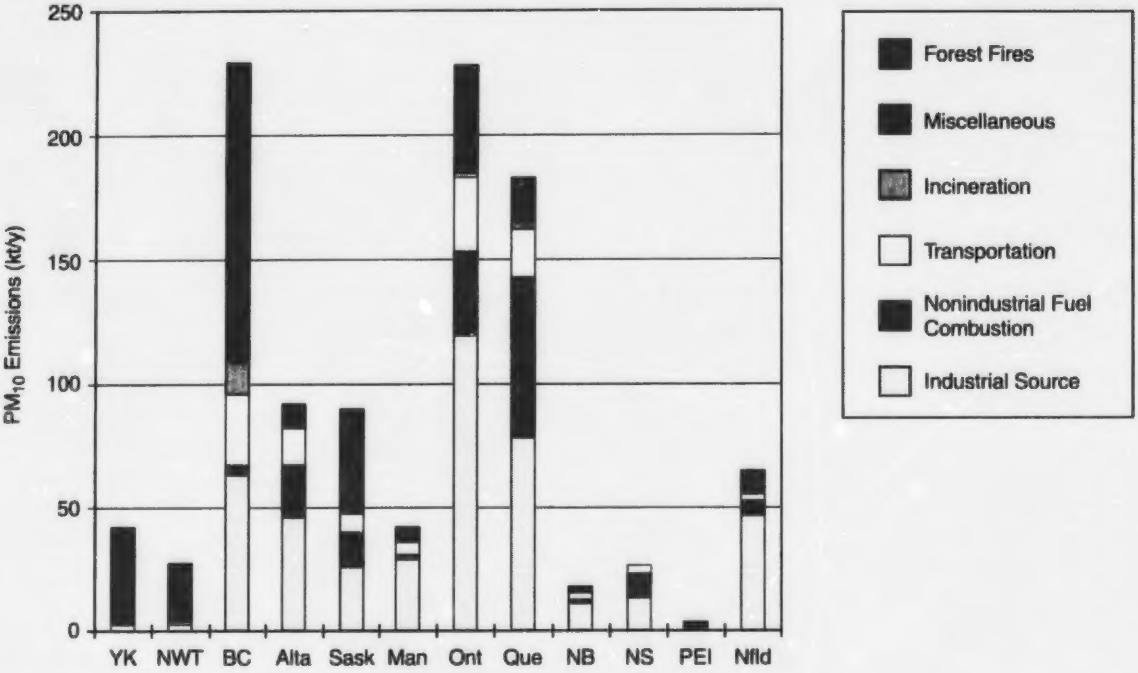
Figure 4.1 Summary of PM_{10} and $PM_{2.5}$ Contributions, 1990. Open sources and secondary particles are not included.

smaller contributions from nonindustrial fuel combustion (16%), transportation (13%), incineration (2%) and miscellaneous sources (1%).

Provincial estimates of PM_{10} emissions for 1990 are summarized in Figure 4.2. Nonindustrial fuel combustion (primarily residential wood combustion) is the dominant source in Prince Edward Island. In BC and Saskatchewan, emissions from forest fires comprise the largest percentage of provincial PM_{10} totals, with contributions from industrial sources also significant. Elsewhere, industrial sources are the largest contributors to provincial PM_{10} emissions. The transportation sector is also a large contributor in British Columbia, Alberta, Ontario and Quebec, whereas nonindustrial fuel combustion is a major source in Alberta, Saskatchewan, Ontario, Quebec and Nova Scotia.

Although excluded from the above figures, preliminary estimates show that fugitive dust sources account for a high percentage of PM_{10} emissions in most provinces (Deslauriers, 1996b). This agrees with findings in the US, where emissions from fugitive dust and miscellaneous sources are estimated to be a factor of 16 higher than emissions from industrial and transportation sources (US EPA, 1994b). Unpaved and paved roads and construction/mining and quarrying sites account for more than 70% of these emissions. In Canada, an estimated 65 Mt of total PM is attributed to open sources (Deslauriers, 1996a), of which more than 90% is from roads and construction sites. These sources are generally episodic in nature, being highly dependent on meteorological conditions such as wind speed and precipitation. For example, 24-hour emission estimates for a day in which wind gusts exceeded

Figure 4.2 Summary of Provincial PM_{10} Emissions, 1990. Open fugitive sources and secondary particles are not included.



96 km/h accounted for 20% of annual fugitive dust emissions in the Coachella Valley, CA (South Coast Air Quality Management District, 1994). As a result, fugitive dust emission estimates exhibit considerable year to year variability. Some concerns have been raised regarding the validity of emission inventory estimates for fugitive dust emissions, owing to discrepancies with estimates obtained from receptor modeling studies. For example, Lowenthal et al. (1994) report that fugitive dust emissions based on inventory estimates accounted for 85% of total PM_{10} emissions in the South Coast Air Basin (SoCAB) of Southern California. In comparison, geological contributions (including road dust, natural soils, agricultural soils and construction material) based on a receptor modeling study in the SoCAB ranged from 15-30% in the summer and from 10-17% during the fall (Watson et al., 1994b).

Also, excluded from aforementioned emission estimates are secondary particles. Unlike fugitive dust emissions, secondary particles are predominantly found in the fine fraction. Limited studies in the Lower Fraser Valley indicate that secondary particles (sulphates and nitrates) comprise up to approximately 50% of $PM_{2.5}$ in parts of the region during the summer months (Pryor and Steyn, 1994a). A source apportionment study in downtown Toronto (Lowenthal, 1997) shows that secondary sulphate and nitrate contribute 33% and 6%, respectively, to $PM_{2.5}$ concentrations measured at the Evans Street site between August-October 1995. In a review of several receptor modeling studies in the western US, Lowenthal (1997) found that secondary sulphate contributions to $PM_{2.5}$ ranged from 1.2% in Phoenix, AZ to 41.8% in Steubenville, OH. Secondary nitrate contributions ranged from undetectable levels in Steubenville, OH to 49.5% in Rubidoux, CA.

Emission estimates for the precursor species SO_2, NO_x and VOCs are included in national and provincial inventories and summarized in Table 4.3 (based on Deslauriers, 1996a). Similar estimates for NH_3 are not available. Precursor emission rates should not be misconstrued as being directly proportional to secondary particle production. As discussed in Section 2.7, secondary particles are the product of various chemical and physical transformations taking place in the atmosphere that may be highly nonlinear.

Table 4.3 Emission of Common Pollutants for Canada 1990 (from Deslauriers, 1996a)

Category/Sector	Emissions in Tonnes		
	SO_2	NO_x	VOC
Industrial Sources			
Abrasive Manufacture	3,490	214	1,758
Aluminum Production	31,967	3,173	834
Asbestos Production	1,748	895	45
Asphalt Production	434	260	2,206
Bakeries		4	3,459
Cement and Concrete Manufacture	31,171	29,372	186
Clay Products	164	292	26
Coal Industry	2,857	1,824	2,457
Crude Oil Production	67,187	2,952	4,390
Ferrous Foundries	1,875	30	1,106
Grain Industries		10	1
Iron and Steel Production	69,514	29,069	27,294
Iron Ore Mining and Beneficiation	45,978	6,809	562
Mining and Rock Quarrying	3,854	3,243	142
Natural Gas Processing	247,532	117,489	3,664
Nonferrous Mining and Smelting	1,401,425	54,594	329
Oil Sands	148,211	15,855	30
Other Chemicals	13,740	15,102	2,818
Other Petroleum and Coal Products	557	458	116
Paint & Varnish Manufacturing	1	9	1,236
Petrochemical Industry	3,291	12,990	28,662
Petroleum Refining	132,647	33,466	82,264
Plastics & Synthetic Resins Fabrication	424	354	12,477
Pulp and Paper Industry	141,717	61,388	20,165
Upstream Oil & Gas Operations		3,159	541,874
Wood Industry	3,479	5,419	15,958
Other Industries	66,665	85,322	88,575
Category total:	**2,419,926**	**483,751**	**842,836**
Nonindustrial			
Commercial Fuel Combustion	19,368	24,192	1,055
Electric Power Generation	690,201	252,356	2,280
Residential Fuel Combustion	29,245	34,971	2,616
Residential Wood Combustion	1,119	5,462	250,526
Category total:	**739,933**	**316,982**	**256,478**

continued

Table 4.3 Emission of Common Pollutants for Canada 1990 (from Deslauriers, 1996a) – *continued*

	Emissions in Tonnes		
Category/Sector	**SO_2**	**NO_x**	**VOC**
Transportation			
Aircraft	1,332	18,506	7,252
Heavy-duty Diesel Vehicles	32,334	324,277	36,555
Heavy-duty Gasoline Trucks	173	11,943	11,445
Light-duty Gasoline Trucks	3,786	95,323	137,294
Light-duty Gasoline Vehicles	9,378	294,501	441,800
Light-duty Diesel Trucks	2,323	2,610	1,088
Light-duty Diesel Vehicles	2,408	1,959	679
Marine	48,410	47,346	32,174
Motor Cycles	19	540	3,023
Off-road Use of Diesel	17,271	271,643	27,763
Off-road Use of Gasoline	1,498	47,153	95,747
Propane Powered Vehicles	7	2,803	1,674
Railroads	14,317	134,143	6,599
Tire Wear			815
Category total:	**133,255**	**1,252,748**	**803,908**
Incineration			
Other Incineration	2265	2659	2594
Wood Waste Incineration	487	4882	52898
Category total:	**2,751**	**7,541**	**55,492**
Miscellaneous			
Application of Surface Coatings		4	177,520
Cigarette Smoking			
Dry Cleaning		1	11,985
Fuel Marketing			96,654
General Solvent Use		9	289,888
Marine Cargo Handling Industry			1
Pesticides and Fertilizer Application		746	42,113
Structural Fires		514	2,376
Category total:		**1,275**	**620,538**
TOTAL FOR CANADA	**3,295,867**	**2,062,297**	**2,579,051**

*The Carbon Black Sector was grouped under the Other Industries Sector to protect confidentiality of information.

In 1990, an estimated 3.2 Mt of SO_2 were discharged from anthropogenic sources in Canada (Deslauriers, 1996a). The largest contributor was the industrial and manufacturing sector, which was responsible for approximately 70% of SO_2 emissions. Emissions from copper and nickel smelting alone accounted for 40% of these emissions.

NO_x emissions are a by-product of combustion activities. In 1990, an estimated 2.1 Mt of NO_x was emitted from anthropogenic sources in Canada. Transportation accounted for 60% of this amount, and stationary fuel combustion an additional 16%.

Anthropogenic VOC emissions result from the incomplete combustion of fuel and from evaporative losses. Approximately 2.6 Mt of VOCs were discharged in Canada in 1990. Industrial sources (predominantly the oil and gas sector) and the transportation sector respectively accounted for 33% and 31% of this amount. Miscellaneous sources such as fuel marketing, solvent use and the application of surface coatings accounted for a further 24%.

4.5 UNCERTAINTIES IN EMISSION ESTIMATES

Uncertainties in emission inventories may be introduced through several ways, including

- partial inclusion of all sources,
- inappropriate emission factors applied,
- temporal variations in sources not considered (e.g. activity rates assumed to be constant with time) and
- poor characterisation of control efficiencies.

Although uncertainty estimates are unavailable for the 1990 PM_{10} or TSP inventory, estimates were made for 1990 CO, NO_x and SO_x emissions (McCann, 1995). These estimates were based on a mix of classical methods and scientific judgment. Calculated uncertainties are as shown in Table 4.4. Uncertainties vary across individual source sectors, and from sector to sector. For example, NO_x emissions from electrical generation were generally based on stack testing, flue gas rate measurements and analytical techniques. The associated uncertainty was estimated to be ±8%. In contrast, emissions from area fuel sources, which were based on US EPA emission factors, were estimated to have an uncertainty of ±50%. Even greater uncertainties can be expected for emission estimates for open sources, due to the very limited amount of published research for these emission factors (Deslauriers, 1996a).

Table 4.4 Estimated NO_x, SO_x and CO Uncertainties (from Deslauriers, 1996a)

Pollutant	Current Data Total	Percentage Uncertainty
SO_x		
All Sectors	3,235	± 5
NO_x		
Non-Transportation Sector	802	*
MOBILE5C	718	*
Other Gasoline User	47	*
Other Transport (c)	459	± 30
Total NO_x	2,026	±20
CO		
Non-Transportation Sector	2,651	± 30
MOBILE5C	5,645	±25
Other Gasoline User	1,441	±20
Other Transport (c)	*	
Total CO	10,004	±30

(a) Excludes miscellaneous categories, forest and other fires.

(b) Confidence levels 95% for SO_x, and NO_x, 85% for CO.

(c) 3,000 tonnes due to taxis and propane vehicles not included.

*could not be determined.

4.6 SUMMARY

Particle emissions result from both natural and anthropogenic activities. Examples of natural sources include sea salt, windblown dust, volcanic emissions, vegetation and wildfires. Important anthropogenic sources include the industrial sector, nonindustrial fuel combustion, prescribed burning, and transportation. Particles may be discharged directly into the atmosphere (primary) or formed in the atmosphere through various chemical and physical transformations (secondary).

The size and chemical composition of the particles formed will reflect the parent materials and the formation processes involved. High-temperature combustion processes and secondary reactions will yield particles predominantly in the fine fraction. Natural and anthropogenic sources involving mechanical processes contribute more greatly to the coarse fraction. The physical and chemical characteristics of particle emissions from various sources were described.

National emission estimates for primary PM_{10} emissions and contributions to the fine and coarse fractions were presented. Excluding open fugitive dust sources, an estimated 1.2 Mt of PM_{10} was emitted directly to the atmosphere in 1990. The fine fraction accounted for almost three-quarters of this amount. On a national scale, significant sources of PM_{10} include industrial sources (36%), biogenic sources (24%), prescribed burning (15%), nonindustrial fuel combustion (13%) and transportation (9%). $PM_{2.5}$ contributions show a similar breakdown, except that the portion from biogenic sources rises to 28% and that from industrial sources decreases to 29%. Preliminary estimates show that emissions from open fugitive dust sources such as paved and unpaved roads, construction sites and agricultural operations account for a high percentage of PM_{10} emissions in most provinces. Limited studies in the Lower Fraser Valley of British Columbia show that secondary particles may account for a significant fraction of $PM_{2.5}$ during the summer months.

5 AMBIENT LEVELS OF PARTICULATE MATTER

Canadian PM data are typically collected over a 24 h averaging period on a one-day-in-six sampling regime. By operating on this schedule, given a long enough sampling period, each day of the week is equally well sampled, and hence all conditions during the week are represented. This chapter focuses primarily upon the 24 h data because it is most abundant and has formed the basis of most of the analyses in the epidemiological research.

5.1 BACKGROUND CONCENTRATIONS OF PM

Natural sources contribute to both fine and coarse particles in the atmosphere. 'Background' PM is generally defined as the distribution of PM concentrations that would be observed in the absence of anthropogenic emissions of PM and precursor emissions of VOC, NO_x, and SO_x in North America (US EPA, 1995).

Background levels of PM vary by geographic location and season. The natural component of background arises from: physical processes of the atmosphere that entrain small particles of crustal material (i.e., soil) and emissions of organic particles and nitrate precursors as a result of natural combustion sources (e.g., wildfires). Vegetation also emits fine organic aerosols and vapour phase precursors. Volcanoes and biogenic sources also emit sulphate precursors. The magnitude of the natural portion of PM for a given location is difficult to determine because of the influence of long-range transport of anthropogenic particles and precursors. The US EPA PM Criteria Document provides the estimates of regional PM_{10} and $PM_{2.5}$ background for annual, or longer averaging times, shown in Table 5.1. The lower bounds of the estimates are based on compilations of natural versus human-made emission levels, ambient measurements in remote areas, and regression studies using human-made and/or natural tracers (NAPAP, 1991; Trijonis, 1982; Malm et al., 1994). The upper bounds are derived from multi-year annual averages of the "clean" remote monitoring sites in the IMPROVE network (Malm et al., 1994) and reflect the effects of both background and anthropogenic emissions from within North America.

Table 5.1 PM_{10} and $PM_{2.5}$ Regional Background Levels (United States)

	Western US ($\mu g/m^3$)	Eastern US ($\mu g/m^3$)
PM_{10}, annual average	4 - 8	5 - 11
$PM_{2.5}$, annual average	1 - 4	2 - 5

The components of background PM as determined by Trijonis et al., (1990) are shown in Table 5.2. The eastern US is estimated to have more natural, organic fine particles and more water associated with hygroscopic fine particles than the west.

The range of expected background concentrations on a short-term basis is much broader. Specific natural events such as wildfires, volcanic eruptions, and dust storms can lead to very high levels of PM comparable to or greater than those observed in polluted urban atmospheres. Excluding extreme events, upper bound estimates of daily background as high as 12 $\mu g/m^3$ PM_{10} have been made based on short-duration studies in remote "clean" areas of the eastern US (Wolff et al., 1983). Based on 24 h peak to annual mean ratios for $PM_{2.5}$ data taken from four southeastern IMPROVE sites (Bachmann, 1996), EPA estimates that the highest background 24 h $PM_{2.5}$ levels over the course of a year could be 4 to 6 times higher than the annual average (i.e., on the order of 15 to 20 $\mu g/m^3$).

5.2 AVAILABLE MONITORING DATA

The majority of PM measurements in Canada have been made using hi-vol (TSP) or tape sampler (CoH) instruments. Manual measurements of PM_{10} using size selective hi-vols (SSI) and dichotomous samplers and manual measurements of $PM_{2.5}$ using dichotomous samplers began in 1984. Continuous instruments (Tapered Element Oscillating Microbalance or TEOM samplers) that have recently been installed at various sites throughout British Columbia and other regions of Canada are designed to provide hourly average estimates of PM_{10} mass. The data

Table 5.2 Components of Background PM

	Annual Average concentration ($\mu g/m^3$)		
	East	West	Error factor
Fine sulphates (as NH_4HSO_4)	0.20	0.10	2
Fine organic compounds	1.50	0.50	2
Fine elemental carbon	0.02	0.02	2-3
Fine ammonium nitrate	0.10	0.10	2
Fine soil dust	0.50	0.50	1.5-2
Water associated with the fine particles	1.00	0.25	2
Coarse particles	3.00	3.00	1.5-2
TOTAL "estimated natural background" PM_{10} (sum of the levels quoted by Trijonis 1990)	6.32	4.47	

Notes:

(1) Eastern USA is defined by Trijonis et al., (1990) as "the area up to one tier of states west of the Mississippi." Western USA includes "the mountain/desert regions of the western United States."

(2) "Fine" refers to aerosols of 2.5 μm in diameter.

(3) "Coarse" particles have diameter 2.5-10 μm.

records from these instruments are too short to provide much information regarding variability of PM_{10} concentrations on time frames of months or years. They do provide insight into short-term variability of PM_{10}, which may play an important role in determining personal exposure, health impacts, and information regarding source apportionment of PM_{10}.

5.2.1 National Air Pollution Surveillance (NAPS) Network

The NAPS network is a federal/provincial/municipal co-operative program that compiles data from all government monitoring programs across Canada. Data is stored in a unified database. In 1994, 93 provincial and municipal TSP monitoring sites and 41 CoH monitoring sites were included in the database. The CoH measurement sites were located in the cities of Montréal, Edmonton, Calgary and Vancouver and in the province of Ontario. The TSP data are collected over 24 hours every sixth day (on a unified national sampling schedule) whereas the CoH instruments are operated continuously and hourly averaged data are reported to the data base.

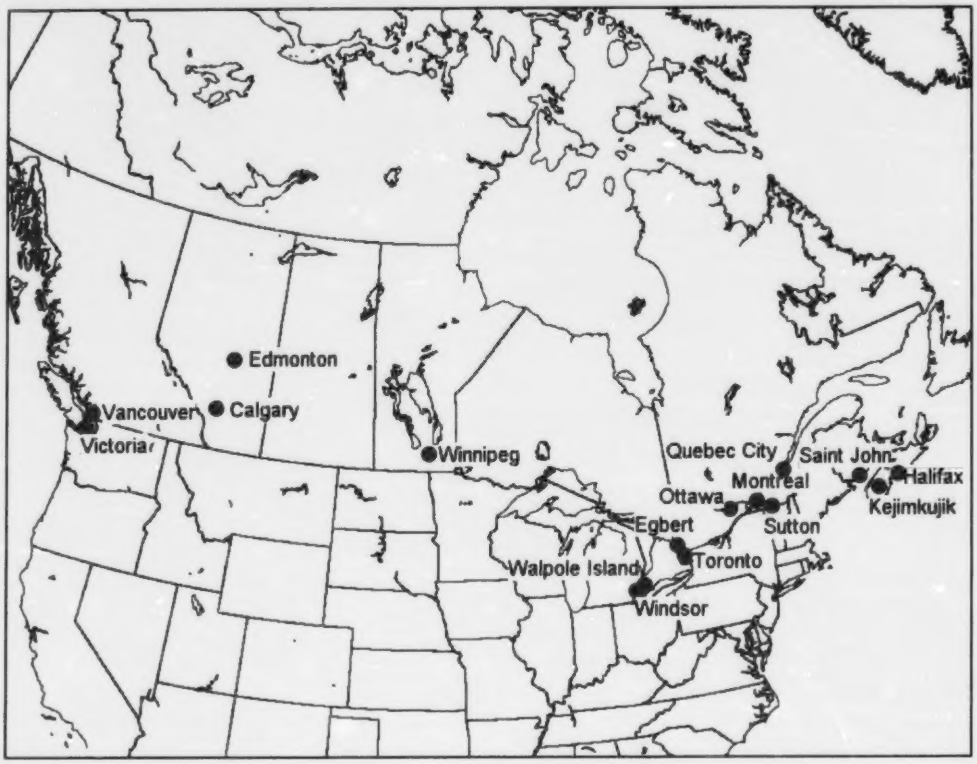
A national PM_{10} and $PM_{2.5}$ monitoring program has been in operation since 1984. Site details and dates of operation are shown in Table 5.3 and site locations are given in Figure 5.1. In 1984, there were 11 sites operating and in 1995, there were 24 samplers in operation. Data has only been collected at four rural locations (Kejimkujik, Sutton, Egbert and Walpole Island), the remainder being urban. The Walpole Island site is very close to metropolitan Detroit (25 km) and is influenced by nearby agricultural operations. Most sites are equipped with dichotomous samplers and report both fine (< 2.5 μm) and coarse (2.5 to 10 μm) mass results. The sum of fine and coarse represents PM_{10}. Some sites report only PM_{10} using SSI samplers. Samples are typically collected over 24 h sampling periods once every six days, although a number of eastern sites were operated daily during the summers of 1993 to 1995 as part of the Canadian Acid Aerosol Monitoring Program (CAAMP). Most dichotomous filter samples are analyzed by energy dispersive X-ray fluorescence (EDXRF) for approximately 50 elements and by high performance liquid chromatography (HPLC) for a number of anions and cations. Beginning in 1994, hourly PM_{10} data from TEOM instruments at NAPS sites are being reported to the database.

Table 5.3 PM$_{10}$ and PM$_{2.5}$ Monitoring Locations and Available Data - National Network

NAPS ID	City	Address	Site Type	Available Data			Sampler Type
				Start Date	End Date	N	
10101	St. John's	Duckworth/Ordinance	Urban	21-Sep-84	16-Oct-85	39	D
40201	Saint John	110 Charlotte St.	Urban	04-Jan-88	28-Dec-94	268	S
40203	Saint John	Forest Hills	Suburban-Industrial	23-Jun-92	11-Nov-95	819	D,S
40204	Saint John	Police Station	Suburban	13-Jan-93	28-Dec-94	106	S
40205	Saint John	Regional Centre	Urban	01-Jan-93	31-Jul-94	85	S
30311	Sydney	Whitney Pier Fire Stn.	Urban-Industrial	04-Jan-88	28-Dec-94	269	S
30101	Halifax	N.S. Technical College	Urban	06-May-84	01-May-94	315	D
30118	Halifax	1657 Barrington St.	Urban	02-May-94	23-Dec-95	272	D
30119	Dartmouth	Harbor View School	Urban	10-Oct-93	03-Apr-95	83	D
30501	KejimkujiK	National Park	Rural	08-Jul-92	17-Dec-95	506	D
50104	Montreal	1125 Ontario Est	Urban	06-May-84	06-Oct-95	815	D
50109	Montreal	Duncan/Decarie	Urban-Roadway	03-Sep-84	21-Jan-95	367	D
50307	Quebec City	Parc Cartier Breboeuf	Urban	13-Apr-85	23-Oct-94	265	D
50308	Quebec City	Des Sables	Urban-Industrial	22-Nov-94	06-Sep-95	40	D
54101	Sutton		Rural	08-May-93	28-Sep-93	136	D
60104	Ottawa	Rideau/Wurtemburg	Urban	06-May-84	29-Dec-95	470	D,S
60204	Windsor	471 University Ave.	Urban	20-Jul-87	08-Apr-94	360	D
60211	Windsor	College/Prince	Urban	29-May-90	25-Aug-95	615	D
60417	Toronto	26 Breadalbane Street	Urban	12-May-84	17-May-90	272	D
60424	Toronto	Bay & Grosvenor	Urban	12-Jun-92	06-Sep-95	597	D
60403	Toronto	Evans & Arnold	Urban-Roadway	01-May-95	29-Dec-95	96	D
60512	Hamilton	Elgin & Kelly	Urban-Industrial	01-May-94	17-Dec-95	329	D
61901	Walpole Island		Rural	22-Jan-88	04-Mar-95	329	D
64401	Egbert		Rural	19-May-93	18-Oct-95	304	D
70119	Winnipeg	65 Ellen Street	Urban	06-May-84	11-Dec-95	550	D
80110	Regina	2505 11th Ave.	Urban	04-Jan-88	28-Dec-94	409	S
80209	Saskatoon	Idylwyld Dr./33rd. St.	Urban	06-Oct-88	02-Nov-92	217	S
80211	Saskatoon	511 1st. Ave. N.	Urban	08-Nov-92	28-Dec-94	121	S
90130	Edmonton	10255-104th Street	Urban	30-May-84	29-Dec-95	497	D,S
90204	Calgary	316-7th Ave.	Urban	28-Aug-84	27-Apr-87	144	D
90227	Calgary	611-4th Street	Urban	03-May-87	29-Dec-95	472	D
100106	Vancouver	2294 West 10th Ave.	Urban	06-May-84	17-Jan-86	108	D
100111	Vancouver	Rocky Pt. Park	Urban	28-Aug-84	17-Nov-95	442	D,S
100118	Vancouver	2550 West 10th Ave.	Urban	13-Dec-86	17-Dec-95	296	D,S
100109	Vancouver	970 Burrard	Urban	25-Sep-89	31-Jul-94	246	S
100303	Victoria	1250 Quadra St.	Urban	13-Jan-85	29-Dec-95	508	D

*D - Dichotomous Sampler, S - Size Selective Inlet Hi-vol Sampler

Figure 5.1 Location of Monitoring Sites – National PM_{10} and $PM_{2.5}$ Monitoring Network



5.2.2 British Columbia Network

The network of PM_{10} observing sites in British Columbia is dense: about 80 sites in the province have recorded 24 h average PM_{10} levels during the last decade using SSI samplers (Figure 5.2). Since 1992, collection of 1 h PM_{10} averages using TEOMs has also occurred at a number of sites in the province. The sites with the longest and most complete PM_{10} data records are summarized in Table 5.4. Only data from these sites will be discussed later in this section. A limited number of $PM_{2.5}$ measurements have been made in British Columbia. $PM_{2.5}$ is currently monitored at sites in Prince George, Quesnel, Williams Lake and Chilliwack using various sampling techniques. Short-term $PM_{2.5}$ monitoring studies have previously been conducted in Vernon and Nakusp. Additional $PM_{2.5}$ data are available from ten locations in southwestern BC which were part of the **RE**gional **V**isibility **E**xperimental **A**ssessment in the **L**ower Fraser Valley (REVEAL) study conducted during the summer of 1993 (Sakiyama, 1994). The REVEAL samples were also analyzed for metals, ions and organic and elemental carbon.

Figure 5.2 Map of PM$_{10}$ Monitoring Sites in BC

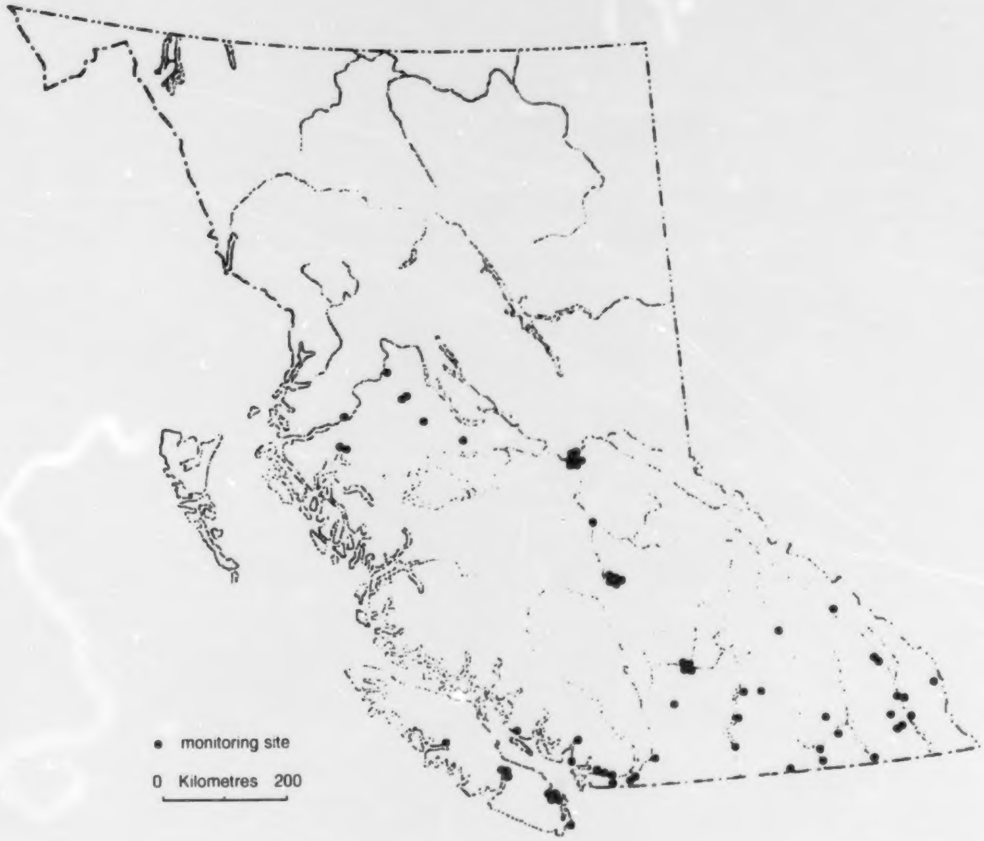


Table 5.4 Locations of PM$_{10}$ Monitors in British Columbia

				Dates of operations		# of samples
Site #	City	Lat	Long	Start	Last sample	
110264	Port Alberni	491401	1244810	6/2/85	24/8/94	389
605001	Kamloops	504220	1202636	5/1/90	27/12/93	214
E206112	Williams Lake	not given	not given	20/5/87	30/8/94	207
E206113	Quesnel	not given	not given	1/11/90	30/8/94	185
E206169	Pitt Meadows	491232	1224223	17/2/91	29/9/84	177
E206241	Cranbrook	493021	1154553	19/5/85	31/3/91	155
E206304	Kelowna	495138	1192833	1/9/89	20/4/94	234
E206589	Smithers	544652	1271037	16/7/90	26/4/94	194
E206725	Kamloops	not given	not given	10/2/90	30/8/94	240
E206931	Castlegar	491936	1173949	23/4/90	30/8/94	158
E208805	Weyside	not given	not given	5/5/90	30/8/94	240
E213056	Creston	not given	not given	20/9/90	27/11/94	199
E213114	Chilliwack	not given	not given	1/11/90	8/5/94	198

5.2.3 Ontario Network

The Ontario Inhalable Particulates Network commenced data collection in 1989 at sites in Windsor, Hamilton, Toronto, Thunder Bay and Sault Ste. Marie. Since 1989, the network has expanded and, as of January 1994, incorporates 18 sites at the locations detailed in Table 5.5. PM_{10} data are collected at these sites using SSI samplers and are collected over 24 hours on a one-in-six-day sampling schedule. Eleven TEOM instruments were operating in the province in 1996.

5.2.4 Quebec Network

Monitoring for PM_{10} has been carried out at 17 sites in the province between the years 1991 and 1995. Site locations are provided in Figure 5.3. PM_{10} data are collected at these sites using SSI samplers and are collected over 24 hours on a one-in-six-day sampling schedule.

Table 5.5 Locations of PM_{10} Monitors in the Ontario Inhalable Particulates Network

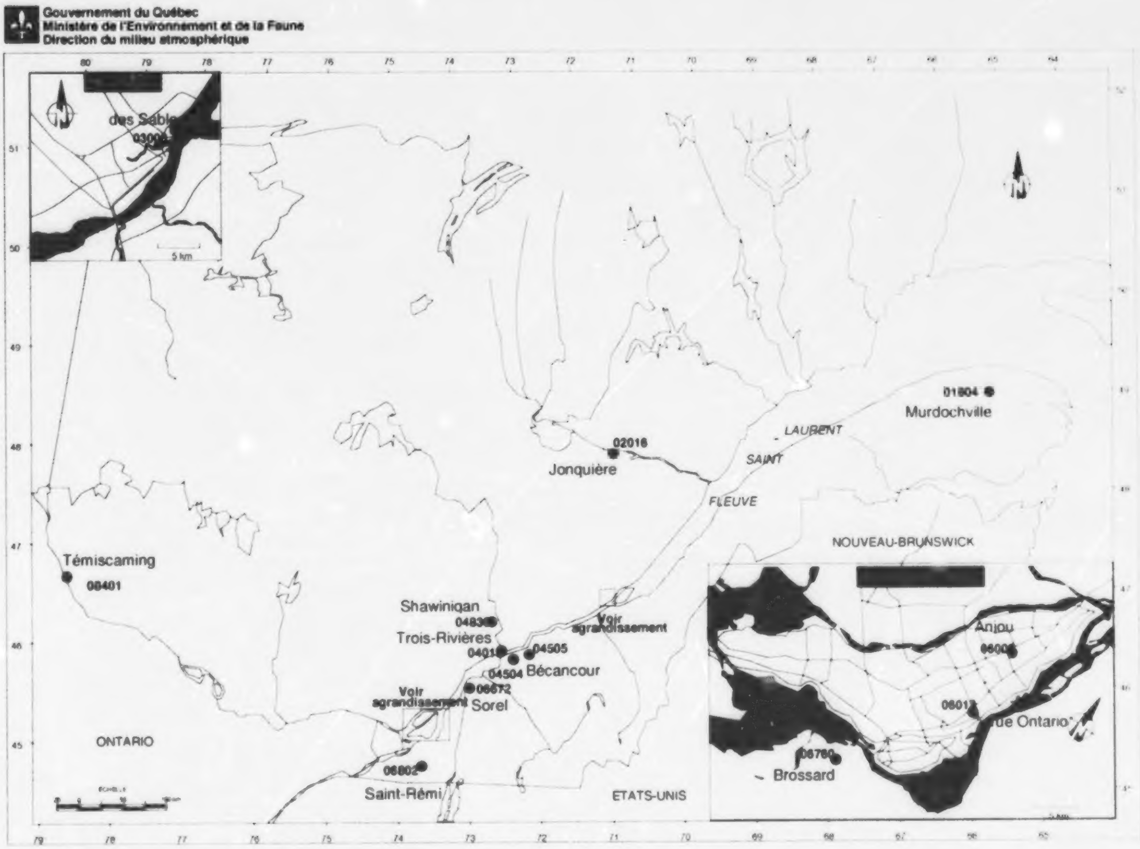
Site #	City	Station name	Lat	Long.	Type	Year
12507	Windsor	Wright/Water St.	42:17	82:16	UI	90
12508	Windsor	467 University Ave. W.	42:19	83:02	C	89
15516	London	801 Commissioners Rd. E.	42:57	81:13	UI	90
22304	Nanticoke	Walpole SPS. Sandusk Rd.	42:50	80:02	RI	92
27308	St. Catherines	King St.	43:10	79:15	C	91
27352	Thorold	185 Queen St., S.	43:07	79:12	UI	90
29300	Hamilton	Elgin/Kelly St.	43:15	79:52	UI	90
29302	Hamilton	Beach Blvd.	43:17	79:47	UI	89
29313	Hamilton	Gertrude/Depew St.	43:15	79:49	UI	90
29324	Hamilton	Buchanan Park P.S.	43:14	79:53	UI	92
31127	Toronto	Bay St./Grovernor Ave.	43:40	79:23	C	89
33127	Scarborough	Kennedy Rd./Lawrence Ave.	43:45	79:16	U	90
35127	Etobicoke	Evans/Arnold Ave.	43:37	79:31	I	90
44127	Oakville	Bronte Rd./Woburn Cres.	43:24	79:44	UI	92
63201	Thunder Bay	615 James St. S.	43:23	89:17	U	89
71342	Sault Ste. Marie	Pumphouse, Bonney St.	46:31	84:23	I	89
71368	Sault Ste. Marie	W.M. Merrifield School	46:32	84:21	UI	90
77326	Sudbury	Lisgar St.	46:29	80:59	UI	91

(1) Year indicates the year data collection commenced.

(2) The sites are designated as U, I, C, UI, RI based upon the environment surrounding the monitoring location.

(3) Key to site designations: U=urban, I=industrial, C=commercial, UI=urban/industrial, RI=rural/industrial.

Figure 5.3 Map of PM_{10} Monitoring Sites in Québec (1996)



5.3 MEASURED PM CONCENTRATIONS IN CANADA

5.3.1 General Characteristics of PM

PM data exhibit a strongly skewed distribution as shown in Figure 5.4 for PM_{10} and $PM_{2.5}$ data from a typical site (Montréal-Duncan/Décarie). The figure shows a histogram and a cumulative frequency distribution of the observations. The data is best fit using a Weibull distribution rather than a lognormal distribution. PM records are dominated by a large number of low values, which can "mask" trends in PM concentrations or the frequency or magnitude of "extreme" events. However, the upper portion of the PM frequency distribution may not be well represented in the data sets from Canadian observing sites because of the limited number of observations available to characterize the extremes of the distribution. The normal sampling frequency for the PM_{10} program has been once every six days. As shown in Table 5.6 the accuracy of the estimate of annual mean concentrations degrades with decreasing sampling frequency (Nehls and Ackland, 1973). The effect of sampling frequency on annual maximum values is provided in Table 5.7 (Nehls and Ackland, 1973). Errors of the estimates of the annual maximum are typically 4 to 6 times larger than the errors in the mean.

Note also that when comparing pollutant statistics or standards from different sampling sites/networks or regulatory agencies, care should be given to ensuring equality of the statistics or standards. Average PM_{10} and $PM_{2.5}$ concentrations are commonly calculated using the arithmetic mean while TSP averages have traditionally been reported using the geometric mean.

PM concentrations exhibit variation on a number of temporal scales: diurnal, hebdomadal (day of week), seasonal and annual (Dann, 1994; Pryor and Steyn, 1994a; Pryor and Barthelmie, 1996). The causes of these variations are multi-faceted and are related to emission variability and variations in geophysical variables such as mixed layer depth, wind speed and humidity levels. Anthropogenic factors affect cycles of pollutant concentrations on a number of time scales, but it is hard to prove causality. If present, the cycle that can most readily be ascribed to anthropogenic causes is the hebdomadal cycle. Given a large enough data set, geophysical conditions should be randomly distributed by day of the week (i.e., no geophysical variable "takes the weekend off"), but emission patterns may be strongly dependent upon the day of the week (Pryor and Steyn, 1995a).

The variability of PM concentrations on diurnal, hebdomadal, seasonal and annual time frames complicates the search for trends within PM time series. However, detection of trends (or the absence of trends) is central to monitoring the success of emission control strategies.

Figure 5.4 Distribution of 24 h PM_{10} and $PM_{2.5}$ Concentrations for a Typical Site (Station 50109 – Montréal)

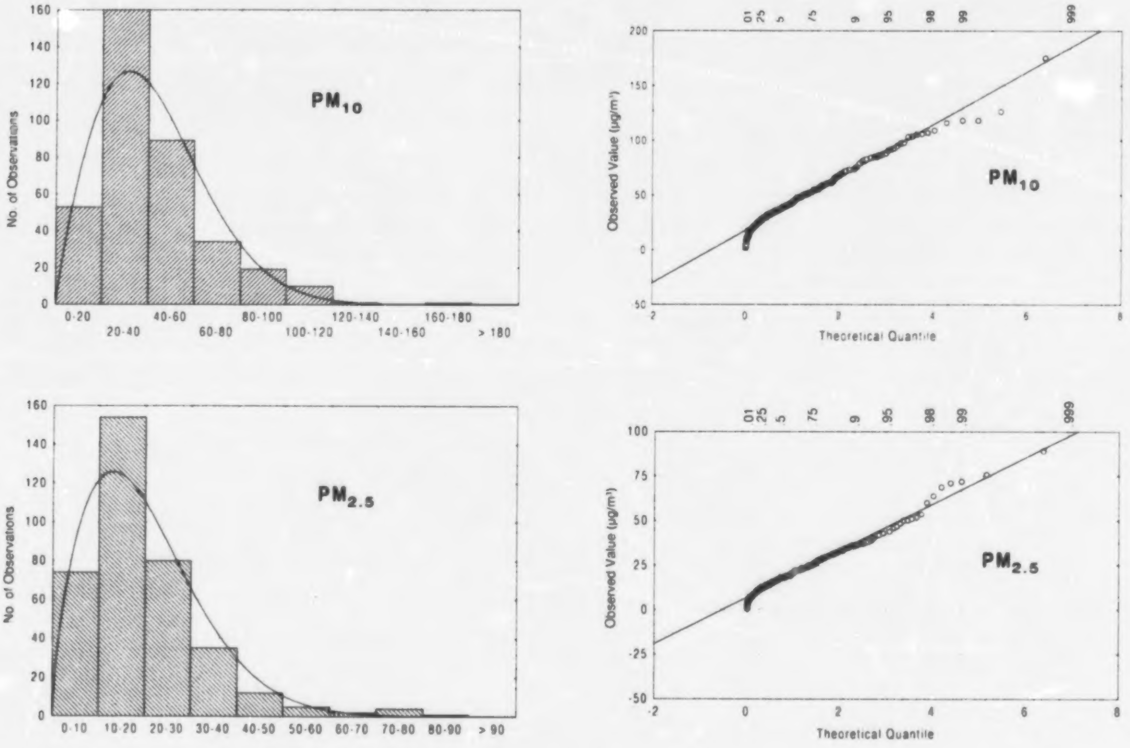


Table 5.6 Error Estimates as a Function of Sampling Frequency

Sampling Frequency (days)	Percent error compared to daily sampling		
	Yearly	Quarterly	Monthly
2	1.4	2.3	3.9
3	1.7	3.2	6.5
4	2.3	4.6	8.7
5	2.7	6.0	10.3
6	3.2	6.5	12.0
7	8.7	10.7	15.1
8	3.8	8.1	14.9
9	5.0	8.9	16.0

Table 5.7 Deviation of the Observed Annual Mean and Maximum fromTrueValues for 42 USTSP Sites

Sampling frequency (days)	Average percentage error (%)	
	Mean	Maximum
1 in 12	7.1	30.1
1 in 6	4.1	22.0
1 in 3	2.1	13.6
1 in 2	1.9	8.9

Notes:

(1) Nehls and Akland (1973) found that the percent error of annual average particulate concentrations calculated based upon data collected every six days compared to that generated from a data set of daily samples was 3.2%.

(2) Dann (1994) estimated that measured and calculated maximum 24 h average PM_{10} concentrations at the NAPS sites differed by up to 61 μgm^{-3} and 88 μgm^{-3} at sites with few data samples (site 60104 in Ottawa and 61901 at Walpole Island, respectively). Sites with more observations showed a smaller discrepancy between calculated and measured maximum 24 h average PM_{10} concentrations (e.g., for data from site 60211 in Windsor the difference was only 16 μgm^{-3}).

5.3.2 TSP and CoH Concentrations

Summary statistics for TSP for NAPS sites for the year 1994 are provided in Table 5.8. Only sites that met annual completeness criteria (at least 8 samples in each quarter of the year) are shown. The highest 24 h value reported was 809 $\mu g/m^3$ at the Yellowknife site. The Calgary industrial site recorded the highest annual geometric mean TSP value of 71 $\mu g/m^3$.

Based on the analysis by Furmanczyk (1994), composite averages of the annual geometric mean TSP concentrations have decreased by 34% between 1981 and 1990. In Ontario, annual geometric mean TSP concentrations have declined by 58% between 1971 and 1994 (OMEE, 1995).

Table 5.9 provides summary statistics for one hour and 24 h CoH values for NAPS network sites for 1994. Maximum 1 h values of 4.7 CoH units were measured at the Toronto-Evans and Arnold site and the Windsor-University Ave. site. A maximum 24 h value of 2.7 CoH units was also recorded at the Toronto-Evans and Arnold site. Most sites recorded 95th percentile hourly values of less than 1.0 CoH unit. Annual means ranged from 0.1 to 0.5 CoH units. There are no NAAQOs for CoH but many Air Quality Index systems have used CoH as a surrogate for PM mass.

Based on trend analysis of NAPS sites (Furmanczyk, 1994), the composite average of station annual means has changed little from 1981 to 1990. For Ontario sites, composite annual means declined from 0.37 CoH units in 1971 to 0.29 CoH units in 1994.

Table 5.8 Summary Statistics for TSP (µg/m³) – 1994 (All NAPS Sites with Complete Data)

Station	C/R/I	City	Location	No. of Samples	Percentiles 10	30	50	70	90	MAX	Geometric Mean	Geometric Std. Dev.	Days > Acc. AQO
10102	C	St. John's	354 Water Street	53	14	18	21	24	30	54	20	1.7	0
20101	C	Charlottetown	56 Fitzroy St.	54	10	14	17	19	27	39	16	1.4	0
30101	C	Halifax	Technical University of N.S.	53	15	20	23	30	40	59	24	1.5	0
30102	R	Halifax	Dalhousie University	54	11	15	17	20	24	31	16	1.5	0
30115	I	Halifax	CFB Shearwater	56	8	12	14	16	18	26	13	1.4	0
40102	C	Fredericton	York Street	61	11	17	22	27	34	55	21	1.6	0
40201	C	Saint John	110 Charlotte Street	50	15	21	26	36	47	51	26	1.6	0
40203	R	Saint John	Forest Hills	41	11	14	21	29	35	40	20	1.6	0
50103	R	Montreal	Pointe-Aux-Trembles	47	19	24	34	38	44	57	31	1.4	0
50104	C	Montreal	1125 Ontario Est	51	27	32	39	49	60	88	39	1.4	0
50109	C	Montreal	Duncan & Decarie	47	26	36	51	65	90	174	50	1.6	2
50110	C	Montreal	Parc Pilon, Mtl.-Nord	48	25	28	32	39	49	57	33	1.3	0
50113	R	Montreal	Chomedey	61	19	25	29	34	40	61	28	1.4	0
50116	R	Montreal	3161 Joseph, Verdun	44	24	33	41	45	66	88	38	1.5	0
50119	R	Montreal	Bourassa	61	22	26	29	32	37	42	28	1.2	0
50120	R	Montreal	4th. Ave. South, Roxboro	51	18	26	31	41	62	96	32	1.6	0
50203	R	Hull	Gamelin & Joffre	58	11	19	25	33	50	73	25	1.7	0
50306	R	Quebec	Gomin	51	15	21	28	36	47	72	27	1.6	0
50307	C	Quebec	Parc Cartier Breboeuf	56	16	26	32	43	63	86	32	1.6	0
50403	C	Sherbrooke	Parc Webster	60	21	28	35	45	55	112	35	1.5	0
50503	C	Chicoutimi	Racine	54	14	24	33	47	73	121	32	1.8	1
50701	C	Sept-Iles	Dequen	46	8	15	21	29	36	83	19	1.8	0
50801	R	Trois-Rivieres	Ursulines	58	17	30	36	46	55	68	34	1.5	0
51201	I	Shawinigan	Frigon	46	25	32	41	53	83	242	43	1.7	1
51302	R	Baie Comeau	39 Ave. Mance	55	9	13	17	21	35	55	17	1.7	0
60101	C	Ottawa	88 Slater St.	60	19	28	33	42	72	172	35	1.6	1
60104	C	Ottawa	Rideau & Wurtemburg	60	15	22	27	36	56	92	27	1.6	0
60203	R	Windsor	9725 Riverside Dr. East	52	26	33	43	51	68	111	42	1.5	0
60204	C	Windsor	467 University Ave. West	53	43	45	54	66	91	136	58	1.4	2
60212	I	Windsor	Wright & Water St.	48	41	52	61	76	101	143	64	1.4	3
60424	C	Toronto	Bay & Grosvenor	60	34	47	58	71	90	137	57	1.5	2
60501	I	Hamilton	Barton & Wentworth	58	32	45	62	78	117	146	60	1.5	5
60503	I	Hamilton	Chatham & Frid	56	37	57	71	88	124	158	68	1.6	6
60511	I	Hamilton	467 Beach Blvd.	55	32	54	72	88	120	183	66	1.6	5

Table 5.8 Summary Statistics for TSP (µg/m³) – 1994 (All NAPS Sites with Complete Data) – *continued*

Station	C/R/I	City	Location	No. of Samples	Percentiles 10	30	50	70	90	MAX	Geometric Mean	Geometric Std. Dev.	Days > Acc. AQO
60512	C	Hamilton	Elgin & Kelly	47	33	46	58	71	120	204	60	1.6	4
60513	R	Hamilton	Vickers Rd. & East 18th. St.	52	23	34	39	51	63	83	39	1.5	0
60605	C	Sudbury	19 Lisgar Street	53	18	25	33	39	55	77	31	1.6	0
60707	I	Sault Ste. Marie	Wm. Merrifield School	56	16	25	34	49	71	107	35	1.8	0
60807	R	Thunder Bay	615 James Street South	52	17	25	32	42	53	70	31	1.6	0
60901	C	London	King & Rectory	52	28	41	53	68	109	147	52	1.7	3
61004	R	Sarnia	Front St. At C.N. Tracks	55	26	34	44	51	72	91	42	1.5	0
61303	C	St. Catharines	71 King Street	50	34	45	53	63	89	104	51	1.4	0
70119	C	Winnipeg	65 Ellen Street	58	19	28	40	49	74	110	37	1.7	0
80102	R	Regina	3211 Albert Street	58	7	12	16	25	40	61	17	1.9	0
80110	C	Regina	2505 11th. Avenue	61	14	24	39	50	70	112	34	1.9	0
80210	R	Saskatoon	1020 Ave I North	57	12	19	26	34	47	72	25	1.7	0
80211	C	Saskatoon	511 1st Avenue North	58	17	26	32	41	54	114	31	1.6	0
90121	I	Edmonton	17 Street & 105 Avenue	58	16	32	46	67	116	198	44	2.1	3
90122	R	Edmonton	127 St. & 133 Avenue	59	14	29	41	52	77	130	38	1.8	1
90130	C	Edmonton	10255 - 104th Street	60	20	32	43	63	97	258	45	1.8	4
90218	I	Calgary	Bonny Brk & 18A St. S.E.	61	43	54	67	86	130	245	71	1.6	8
90222	R	Calgary	39 St. & 29 Ave. N.W.	56	15	21	28	42	56	104	29	1.7	0
90227	C	Calgary	611-4th Street S.W.	60	22	38	47	63	94	301	47	1.7	3
90601	R	Fort Saskatchewan	100 Avenue & 98th Street	57	9	19	24	35	52	73	24	1.8	0
100102	R	Vancouver	100 Richmond Street	53	20	26	34	43	60	91	34	1.6	0
100104	R	Vancouver	4251 Ontario St.	47	14	20	25	32	45	68	25	1.6	0
100110	R	Vancouver	6400 E. Hastings & Kensington	59	10	14	23	29	37	45	20	1.7	0
100111	I	Vancouver	Moody & Esplanade, Port Moody	60	13	17	25	31	40	51	23	1.6	0
100113	I	Vancouver	Annacis Island, Delta	55	18	28	39	55	77	113	39	1.7	0
100114	C	Vancouver	Municipal Hall, Richmond	55	14	20	25	31	39	66	25	1.5	0
100116	R	Vancouver	163 East 48th St., N. Vancouver	55	11	14	19	26	33	41	19	1.6	0
100117	R	Vancouver	3700 Willingdon Ave., Burnaby	54	11	18	27	31	41	67	23	1.7	0
100118	R	Vancouver	2550 West 10th Avenue	54	14	20	25	31	41	66	25	1.5	0
100202	C	Prince George	1011 4th Avenue	60	17	23	35	51	90	174	36	1.9	3
100303	C	Victoria	1250 Quadra St.	52	18	27	34	39	48	61	32	1.5	0
100401	C	Kamloops	301 Seymour St.	61	24	34	41	64	96	134	45	1.7	2
100701	C	Kelowna	1000 Klo Rd.	58	11	22	29	39	69	118	28	1.9	0
119001	C	Whitehorse	Federal Building	51	6	15	22	34	41	82	19	2.1	0
129001	C	Yellowknife	50th Ave. & 49th Street	57	20	37	61	87	232	809	62	2.4	13

Table 5.9 Summary Statistics for Coefficient of Haze (CoH Units per 1000 Lineal Feet) – 1994

Station	C/R/I	City	Location	ID	10	30	50	70	90	95	Max	% Data	Annual Mean	Std. Dev.
50109	C	Montreal	Duncan & Decarie	1 HR	0.0	0.2	0.4	0.7	1.2	1.5	3.6	91.8	0.5	0.5
				24 HR	0.1	0.3	0.5	0.7	1.0	1.2	2.0	91.1		
50110	C	Montreal	Parc Pilon Mtl.-Nord	1 HR	0.0	0.0	0.1	0.2	0.5	0.7	2.1	89.2	0.2	0.2
				24 HR	0.0	0.1	0.1	0.2	0.4	0.5	0.9	89.9		
50115	C	Montreal	1001 Boul Maisonneuve O.	1 HR	0.1	0.2	0.3	0.4	0.7	0.8	3.2	90.0	0.4	0.3
				24 HR	0.2	0.3	0.3	0.4	0.6	0.7	1.4	91.4		
50116	R	Montreal	3161 Joseph Verdun	1 HR	0.0	0.0	0.1	0.1	0.3	0.4	1.2	84.0	0.1	0.1
				24 HR	0.0	0.1	0.1	0.1	0.2	0.3	0.6	83.3		
50120	R	Montreal	4th. Ave. South Roxboro	1 HR	0.0	0.0	0.1	0.2	0.4	0.7	2.4	87.9	0.2	0.3
				24 HR	0.0	0.1	0.1	0.2	0.3	0.5	1.1	86.3		
60104	C	Ottawa	Rideau & Wurtemburg	1 HR	0.1	0.1	0.2	0.3	0.5	0.6	1.8	97.6	0.2	0.2
				24 HR	0.1	0.1	0.2	0.3	0.4	0.5	1.0	97.3		
60204	C	Windsor	467 University Ave. West	1 HR	0.1	0.2	0.2	0.3	0.5	0.7	4.7	98.6	0.3	0.2
				24 HR	0.1	0.2	0.3	0.3	0.5	0.5	2.1	98.1		
60211	R	Windsor	College & South St.	1 HR	0.1	0.2	0.3	0.4	0.6	0.8	4.1	98.6	0.3	0.3
				24 HR	0.2	0.2	0.3	0.4	0.5	0.7	2.1	98.0		
60302	R	Kingston	Napier St.	1 HR	0.1	0.1	0.2	0.3	0.9	1.4	3.6	98.6	0.4	0.4
				24 HR	0.1	0.2	0.3	0.4	0.8	0.9	1.7	97.9		
60403	I	Toronto	Evans & Arnold Ave.	1 HR	0.1	0.3	0.4	0.6	1.0	1.2	4.7	97.7	0.5	0.4
				24 HR	0.2	0.3	0.4	0.6	0.8	0.9	2.7	97.6		
60410	R	Toronto	Lawrence & Kennedy	1 HR	0.1	0.2	0.3	0.4	0.7	0.8	4.5	97.3	0.3	0.3
				24 HR	0.2	0.2	0.3	0.4	0.6	0.7	2.5	97.1		
60413	R	Toronto	Elmcrest Road	1 HR	0.1	0.2	0.3	0.4	0.8	1.1	3.2	97.9	0.4	0.4
				24 HR	0.1	0.2	0.3	0.4	0.7	0.9	1.9	97.4		
60415	R	Toronto	Queensway W & Hurontario	1 HR	0.1	0.2	0.3	0.5	0.8	1.1	3.8	98.5	0.4	0.4
				24 HR	0.2	0.3	0.4	0.5	0.7	0.8	1.6	98.4		
60424	C	Toronto	Bay & Grosvenor	1 HR	0.1	0.2	0.3	0.5	0.7	0.9	2.5	98.2	0.4	0.3
				24 HR	0.2	0.3	0.3	0.5	0.6	0.7	1.5	97.9		
60501	I	Hamilton	Barton & Wentworth	1 HR	0.2	0.3	0.4	0.6	1.1	1.4	4.0	97.7	0.5	0.4
				24 HR	0.2	0.4	0.5	0.6	0.9	1.2	2.3	97.5		
60511	I	Hamilton	467 Beach Blvd.	1 HR	0.1	0.3	0.5	0.8	1.2	1.4	3.6	96.0	0.6	0.5
				24 HR	0.2	0.4	0.6	0.7	1.0	1.1	2.2	95.7		
60512	C	Hamilton	Elgin & Kelly	1 HR	0.1	0.2	0.3	0.5	1.0	1.3	3.7	98.8	0.5	0.4
				24 HR	0.2	0.3	0.4	0.5	0.9	1.1	1.9	98.8		
60513	R	Hamilton	Vickers Rd. & East 18th. St.	1 HR	0.1	0.1	0.2	0.3	0.6	0.8	2.6	98.8	0.3	0.3
				24 HR	0.1	0.2	0.2	0.3	0.5	0.6	1.2	98.8		
60607	R	Sudbury	Science Centre North	1 HR	0.0	0.1	0.1	0.2	0.4	0.5	2.5	92.9	0.2	0.2
				24 HR	0.1	0.1	0.2	0.2	0.3	0.4	0.9	91.7		
60707	I	Sault Ste. Marie	Wm. Merrifield School	1 HR	0.0	0.1	0.2	0.3	0.6	0.9	2.6	91.6	0.3	0.3
				24 HR	0.1	0.2	0.3	0.4	0.5	0.6	1.1	91.4		

Table 5.9 Summary Statistics for Coefficient of Haze (CoH Units per 1000 Lineal Feet) – 1994 – *continued*

Station	C/R/I	City	Location	ID	10	30	50	70	90	95	Max	% Data	Annual Mean	Std. Dev.
60807	R	Thunder Bay	615 James Street South	1 HR	0.0	0.1	0.2	0.3	0.6	0.7	3.3	94.8	0.2	0.3
				24 HR	0.1	0.1	0.2	0.3	0.5	0.6	1.7	94.4		
60901	C	London	King & Rectory	1 HR	0.1	0.1	0.2	0.3	0.5	0.7	3.2	96.1	0.2	0.2
				24 HR	0.1	0.1	0.2	0.3	0.5	0.5	1.7	95.8		
61004	R	Sarnia	Front St. At C.N. Tracks	1 HR	0.0	0.1	0.2	0.3	0.4	0.5	2.4	99.2	0.2	0.2
				24 HR	0.1	0.1	0.2	0.2	0.4	0.4	1.1	99.8		
61201	R	Cornwall	Bedford & Third St.	1 HR	0.0	0.1	0.1	0.2	0.4	0.6	3.8	97.9	0.2	0.2
				24 HR	0.1	0.1	0.1	0.2	0.4	0.5	1.3	97.8		
61302	C	St. Catharines	Argyle Crescent	1 HR	0.1	0.1	0.2	0.3	0.5	0.6	2.7	97.7	0.3	0.2
				24 HR	0.1	0.2	0.2	0.3	0.4	0.5	1.0	97.6		
61502	C	Kitchener	West Ave. & Homewood	1 HR	0.1	0.1	0.2	0.3	0.5	0.6	2.8	99.0	0.3	0.2
				24 HR	0.1	0.2	0.2	0.3	0.4	0.5	1.4	99.0		
61602	R	Oakville	Bronte Rd. & Woburn Cres.	1 HR	0.1	0.2	0.3	0.4	0.7	0.9	2.8	98.7	0.3	0.3
				24 HR	0.1	0.2	0.3	0.4	0.6	0.8	1.7	98.8		
61701	R	Oshawa	Ritson Rd. & Olive Ave.	1 HR	0.1	0.2	0.3	0.4	0.8	1.0	4.1	94.7	0.4	0.3
				24 HR	0.1	0.2	0.3	0.4	0.6	0.8	1.8	94.8		
62001	R	North Bay	O.P.P. Station	1 HR	0.0	0.1	0.1	0.2	0.4	0.6	2.4	91.3	0.2	0.2
				24 HR	0.1	0.1	0.2	0.2	0.4	0.4	0.8	90.1		
64501	R	Fort Frances	Portage/Church St.	1 HR	0.0	0.1	0.1	0.2	0.5	0.6	2.0	98.6	0.2	0.2
				24 HR	0.1	0.1	0.2	0.3	0.4	0.5	0.9	97.8		
70118	R	Winnipeg	Jefferson & Scotia	1 HR	0.0	0.0	0.0	0.1	0.1	0.2	0.9	70.6	---	
				24 HR	0.0	0.0	0.0	0.1	0.1	0.1	0.2	70.9		
70119	C	Winnipeg	65 Ellen Street	1 HR	0.0	0.1	0.1	0.2	0.3	0.3	2.0	80.5	0.1	0.1
				24 HR	0.1	0.1	0.1	0.2	0.2	0.3	0.4	78.6		
90121	I	Edmonton	17 Street & 105 Avenue	1 HR	0.0	0.1	0.1	0.3	0.6	0.9	3.1	99.7	0.2	0.3
				24 HR	0.0	0.1	0.2	0.3	0.5	0.6	1.1	99.6		
90122	R	Edmonton	127 St. & 133 Avenue	1 HR	0.0	0.1	0.2	0.3	0.7	0.9	4.4	98.6	0.3	0.3
				24 HR	0.1	0.2	0.2	0.3	0.6	0.7	2.2	98.2		
90130	C	Edmonton	10255 - 104th Street	1 HR	0.1	0.1	0.2	0.3	0.5	0.7	3.1	99.9	0.3	0.2
				24 HR	0.1	0.2	0.2	0.3	0.4	0.5	0.9	99.8		
90218	I	Calgary	Bonny Brk & 18a St. S.E.	1 HR	0.0	0.1	0.2	0.3	0.6	0.8	4.5	99.5	0.3	0.3
				24 HR	0.1	0.2	0.2	0.3	0.5	0.6	2.2	99.3		
90222	R	Calgary	39 St. & 29 Ave. N.W.	1 HR	0.0	0.0	0.1	0.1	0.2	0.4	2.1	99.8	0.1	0.1
				24 HR	0.0	0.1	0.1	0.1	0.2	0.2	0.8	99.6		
90227	C	Calgary	611-4th Street S.W.	1 HR	0.0	0.1	0.1	0.2	0.4	0.6	2.8	99.2	0.2	0.2
				24 HR	0.1	0.1	0.2	0.2	0.3	0.4	1.5	98.9		
100110	R	Vancouver	6400 E. Hastings & Kensington	1 HR	0.1	0.1	0.2	0.3	0.5	0.6	3.3	90.1	0.2	0.2
				24 HR	0.1	0.1	0.2	0.3	0.4	0.5	0.9	90.0		
100111	I	Vancouver	Moody & Esplanade Port Moody	1 HR	0.1	0.2	0.3	0.4	0.7	0.9	2.0	97.9	0.4	0.3
				24 HR	0.2	0.2	0.3	0.4	0.6	0.7	1.4	98.0		
100112	C	Vancouver	Robson/Hornby	1 HR	0.1	0.2	0.3	0.5	0.7	0.9	2.9	94.6	0.4	0.3
				24 HR	0.2	0.3	0.4	0.5	0.6	0.7	1.5	94.2		

5.3.3 24 h Average PM_{10} Concentrations

National Network (NAPS)

Table 5.10 summarizes the frequency distributions of PM_{10} data from dichotomous and SSI samplers operated at the sites listed in Table 5.3. At the urban sites, with multiple years of data, mean 24 h PM_{10} concentrations range from 9 to 42 µg/m^3 with most sites in the range of 20 to 30 µg/m^3. The highest PM_{10} concentrations (in terms of means and 90th percentiles) were measured at the Montréal-Duncan/Décarie, Windsor University Ave., Windsor College, Hamilton, Walpole Island and Calgary 7th Ave. The three rural sites of Kejimkujik, Sutton and Egbert record mean PM_{10} concentrations of 11, 11 and 17 µg/m^3 respectively, although observations were only available for 1992-1995 for Kejimkujik and Egbert and for May-September, 1993 for Sutton. As noted, relatively high PM_{10} values were recorded at the other rural site at Walpole Island. This may be due to the effect of windblown dust from nearby agricultural operations and due to its proximity to metropolitan Detroit.

In Figure 5.5 selected dichotomous sampler sites are compared in terms of monthly variations in PM_{10} concentrations using a boxplot analysis of medians, 25th and 75th percentiles (inter-quartile range) and non-outlier minimums and maximums. The Montreal and Vancouver/Victoria sites record higher PM_{10} concentrations in the winter months and in particular during January and February. The Ontario sites record the highest concentrations in the summer months with a peak median in August. The Prairie sites experience their highest median concentrations in the spring, presumably due to lack of snow and vegetation cover, higher wind speeds and influence of street-sanding debris. The Maritime sites show variable seasonal variation in PM_{10} concentrations with Saint John and Kejimkujik showing a summer maximum and Halifax a winter maximum. In Figure 5.6, a similar analysis of day of week variations in PM_{10} concentrations is provided. Most urban sites show minimum PM_{10} concentrations on Sunday with maximum concentrations usually occurring during the middle of the week. Using results for all sites, mean midweek (Tuesday/Wednesday) PM_{10} concentrations were 30% higher than Sunday means. For the roadway sites, the differences were larger – up to a 50% increase in PM_{10}. This suggests that there are large day of week differences in anthropogenic emissions and indicates a substantial contribution to PM_{10} from transportation sources.

Yearly variations in PM_{10} concentrations during the 1984 to 1995 sampling period (1995 incomplete for most sites) are examined for the sites in Figure 5.7. There is an apparent decrease in PM_{10} concentrations at most sites with a complete data record with the largest percentage decreases occurring at the Montréal-Duncan/Décarie, Edmonton and Vancouver sites. A trend analysis of PM_{10} data carried out by Dann, 1994 for the period 1984-93 showed a statistically significant ($p < 0.001$) decreasing trend in PM_{10} on a national basis averaging 2% per year. In the US (Curran, 1993), the national composite average PM_{10} concentrations decreased by 17% between 1988 and 1992 (3.4% decrease per year).

The contrast between PM_{10} measurements at the two sites in Montréal (sites 50104 and 50109) and between sites 50109 and 54101 (Sutton) exemplifies one of the problems of monitoring PM_{10}. PM_{10} is not spatially homogeneous. Monitoring site characteristics and the existence (or absence) of significant local sources of PM_{10} may have considerable impact upon the PM_{10} concentration recorded at a given location.

To examine the statistical significance of the variability of PM_{10} concentrations, the chi-squared (χ^2) statistic was calculated for the data conditionally sampled by year, month and weekday. The χ^2 statistic involves comparing an observed distribution with a hypothetical expected distribution. In the current example, a contingency table was constructed for studying the distribution of PM_{10} concentrations (in four "bins": 0–20, 21–40, 41–60, >60 µg/m^3) by year, month and day of the week. A χ^2 test was performed to test the hypothesis that the "columns" (i.e., year/month/day of the week) and "rows" (number of counts in the PM_{10} concentration bin) are not related (Hoel, 1975). In this type of analysis, the χ^2 value produced must be greater than tabulated values of χ^2 at a given significance level for the hypothesis to be rejected. As shown in Table 5.11, the inter-annual variability of the PM_{10} concentrations was significant at greater than the 90% confidence level for all but three of the sites where there were at least 20 observations available for each year.

Table 5.10 Frequency Distributions of 24 h Average PM_{10} Concentrations ($\mu g/m^3$) from Dichotomous Sampler Sites (1984-1995) and SSI Sites (1988-1994) – National Network

				Percentiles							
NAPS ID	City	N	Min.	10	30	50	70	90	Max.	Mean	Std. Dev.
Dichotomous Sampler Sites											
10101	St. John's	39	2	5	12	16	22	29	34	17	8
40203	Saint John	819	1	5	8	12	18	28	70	15	10
30101	Halifax	315	6	12	18	23	29	40	81	25	11
30118	Halifax	272	3	8	11	13	16	22	52	15	7
30119	Dartmouth	83	2	7	9	11	14	19	36	12	6
30501	Kejimkujik	506	1	4	6	8	11	21	60	11	9
50104	Montréal	815	3	11	17	23	30	45	99	26	14
50109	Montréal	367	2	18	28	36	49	74	175	42	24
50307	Québec City	265	2	9	15	18	27	40	71	22	13
50308	Québec City	40	1	5	12	16	20	33	70	18	13
54101	Sutton	136	2	5	7	9	13	21	42	11	7
60104	Ottawa	470	3	8	14	18	24	38	76	21	12
60204	Windsor	360	4	13	20	27	36	55	110	31	18
60211	Windsor	615	5	14	21	27	35	52	105	31	16
60417	Toronto	272	3	13	19	24	32	48	96	28	15
60424	Toronto	597	3	11	16	23	30	47	102	26	15
60403	Toronto	96	6	11	18	25	32	44	63	26	12
60512	Hamilton	329	4	12	19	26	37	54	177	31	19
61901	Walpole Island	329	3	9	16	24	35	61	149	30	23
64401	Egbert	304	1	5	9	13	21	32	77	17	12
70119	Winnipeg	550	3	12	18	24	32	46	112	27	17
90130	Edmonton	497	4	10	16	23	30	45	86	25	14
90204	Calgary	144	6	14	20	27	35	59	114	32	19
90227	Calgary	472	5	10	16	21	29	43	84	24	13
100106	Vancouver	108	8	13	18	25	30	51	84	28	15
100111	Vancouver	442	3	11	17	22	29	43	82	25	13
100118	Vancouver	296	4	9	14	19	23	36	69	21	11
100303	Victoria	508	2	8	11	15	20	30	71	17	10
SSI Sites											
40201	Saint John	268	1	9	15	19	26	38	64	21	11
40203	Saint John	246	1	6	11	14	19	28	59	16	10
40204	Saint John	106	1	3	5	8	11	16	50	9	7
40205	Saint John	85	3	6	9	12	16	25	77	14	10
30311	Sydney	269	5	11	14	18	24	39	111	22	15
60104	Ottawa	405	3	8	12	15	22	32	77	19	11
80110	Regina	409	4	8	13	19	28	48	367	25	24
80209	Saskatoon	217	2	8	13	17	23	35	76	20	13
80211	Saskatoon	121	4	8	12	15	20	28	75	18	11
90130	Edmonton	404	5	12	17	23	33	49	132	28	16
100111	Vancouver	249	5	9	14	17	22	34	53	19	9
100118	Vancouver	183	4	9	12	15	21	30	58	18	10
100109	Vancouver	246	5	12	17	22	28	40	70	24	11

Figure 5.5 Monthly Variation in PM_{10} Concentrations for Selected Monitoring Locations (1984-1995). The Box Plots Indicate the Median, 25th and 75th Percentiles and the Non-Outlier Minimum and Maximum Concentrations.

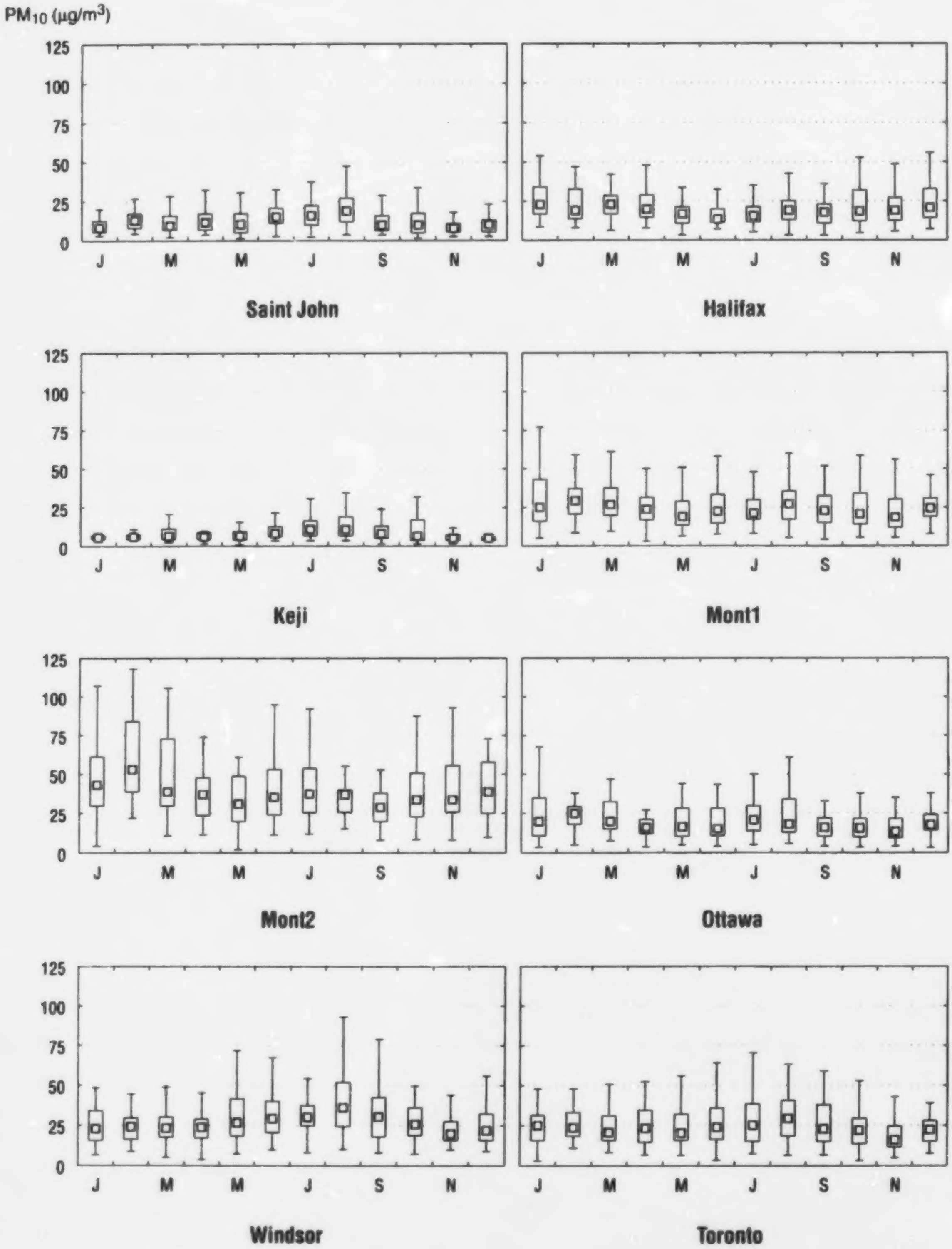
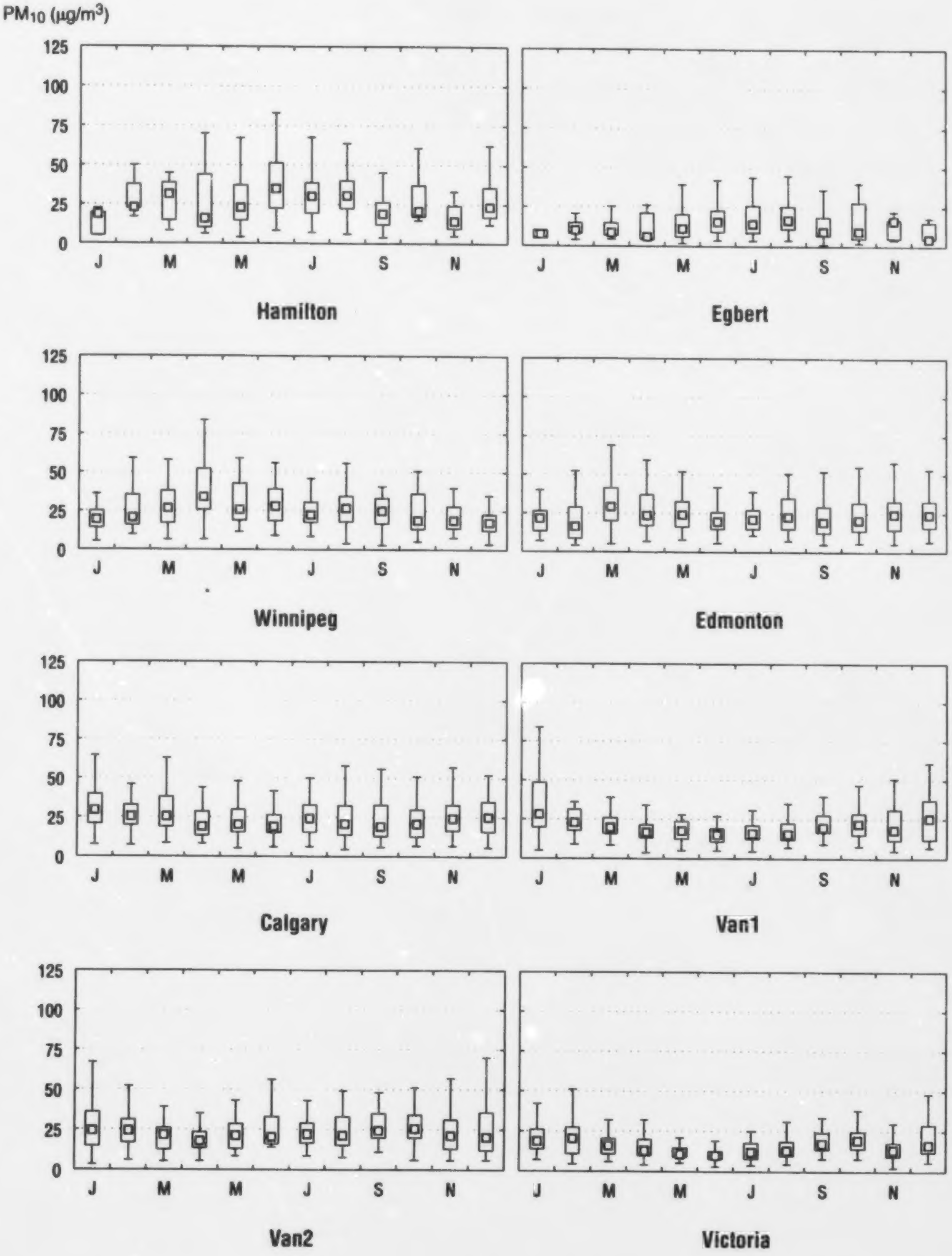


Figure 5.5 Monthly Variation in PM_{10} Concentrations for Selected Monitoring Locations (1984-1995). The Box Plots Indicate the Median, 25th and 75th Percentiles and the Non-Outlier Minimum and Maximum Concentrations. *(continued)*



Hamilton

Egbert

Winnipeg

Edmonton

Calgary

Van1

Van2

Victoria

Figure 5.6 Day of Week Variation in PM_{10} Concentrations for Selected Monitoring Locations (1984-1995). The Box Plots Indicate the Median, 25th and 75th Percentiles and the Non-Outlier Minimum and Maximum Concentrations.

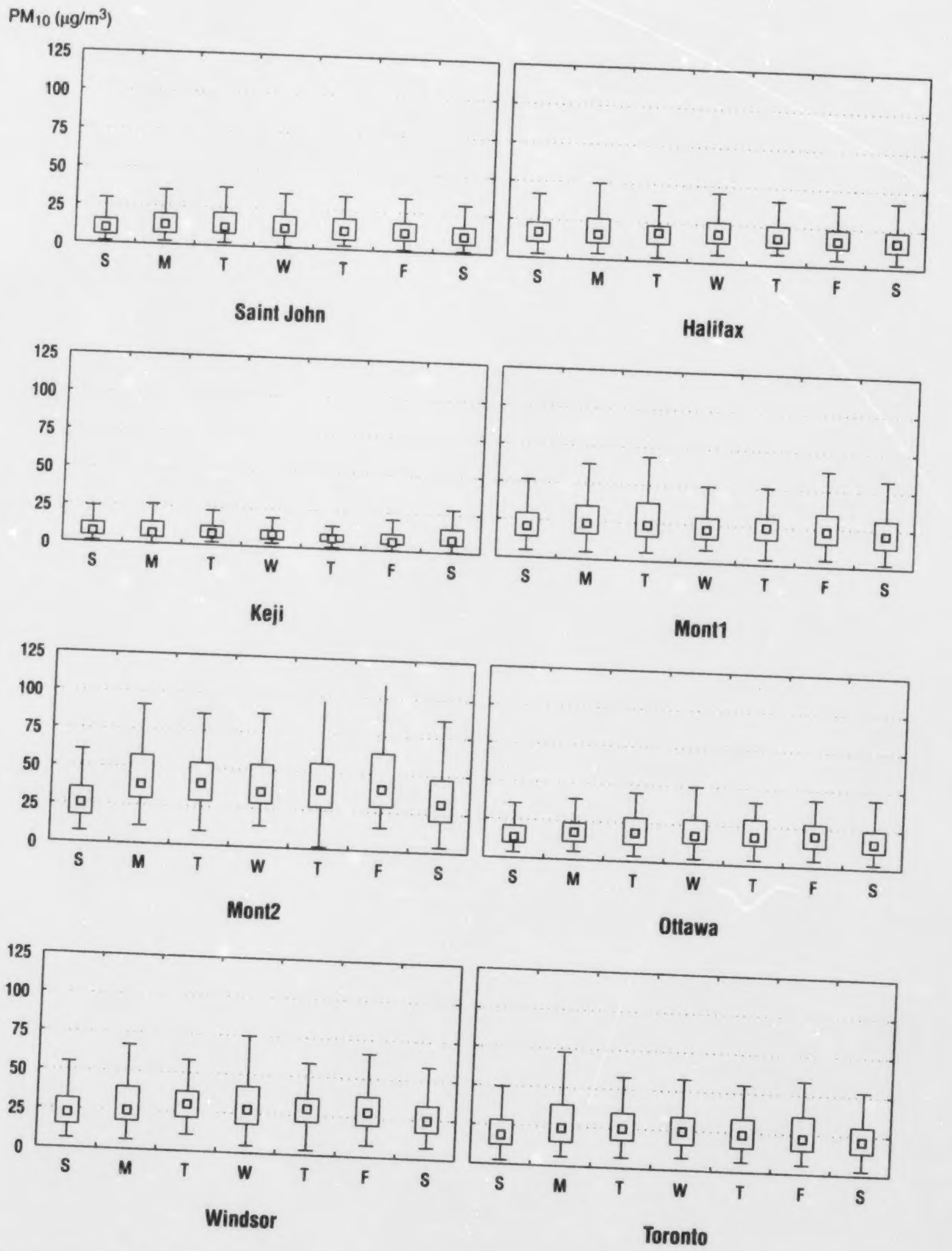
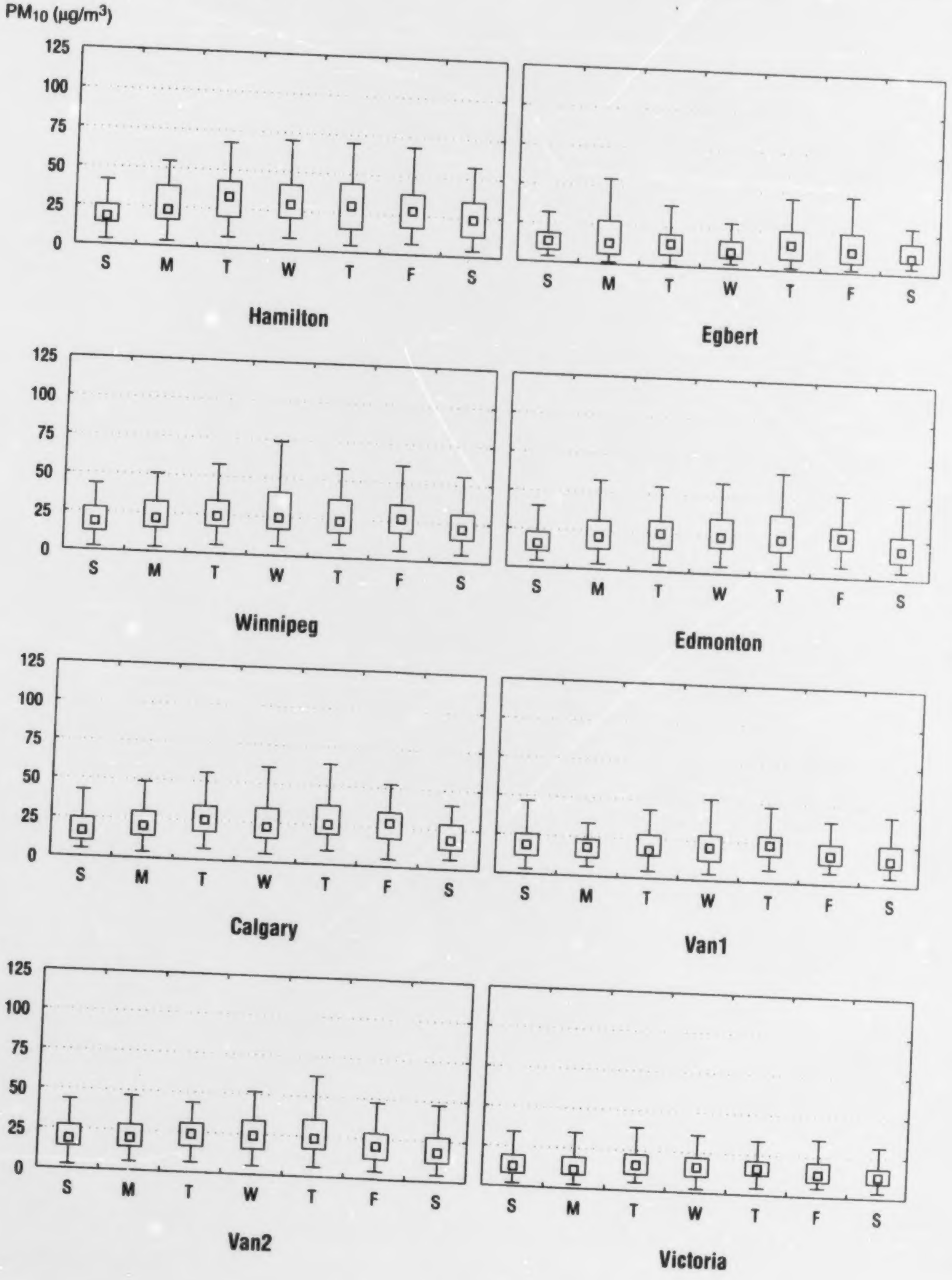


Figure 5.6 Day of Week Variation in PM_{10} Concentrations for Selected Monitoring Locations (1984-1995). The Box Plots Indicate the Median, 25th and 75th Percentiles and the Non-Outlier Minimum and Maximum Concentrations. ***(continued)***



Hamilton

Egbert

Winnipeg

Edmonton

Calgary

Van1

Van2

Victoria

Figure 5.7 Yearly Variation in PM_{10} Concentrations for Selected Monitoring Locations (1984-1995). The Box Plots Indicate the Median, 25th and 75th Percentiles and the Non-Outlier Minimum and Maximum Concentrations.

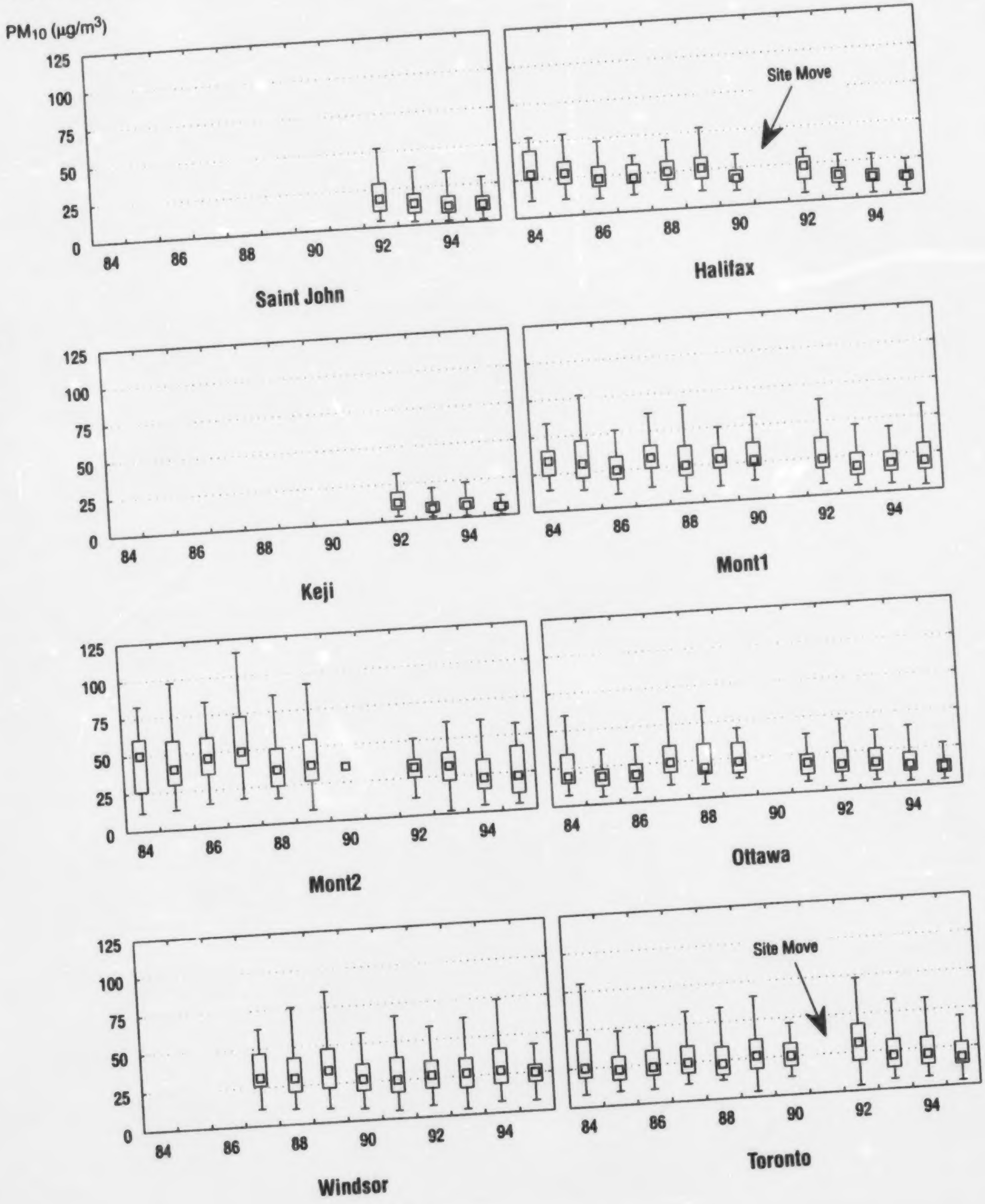
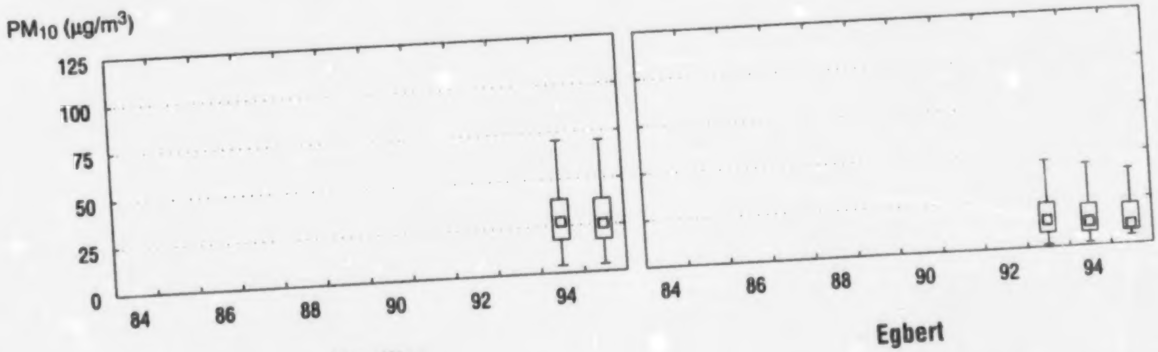
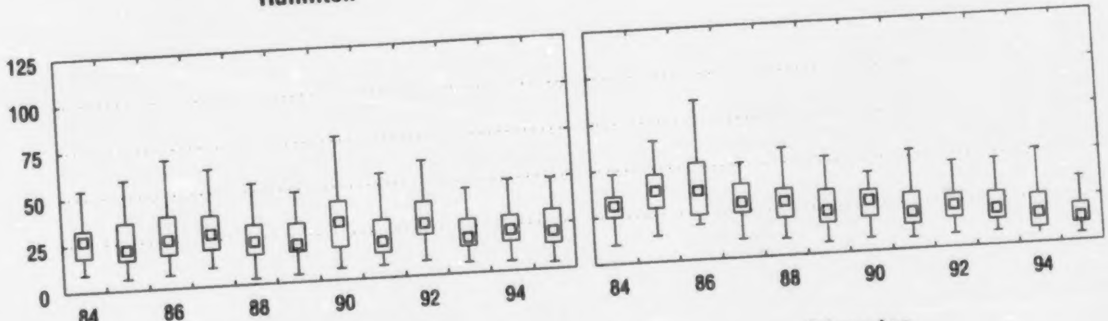


Figure 5.7 **Yearly Variation in PM_{10} Concentrations for Selected Monitoring Locations (1984-1995). The Box Plots Indicate the Median, 25th and 75th Percentiles and the Non-Outlier Minimum and Maximum Concentrations.** ***(continued)***



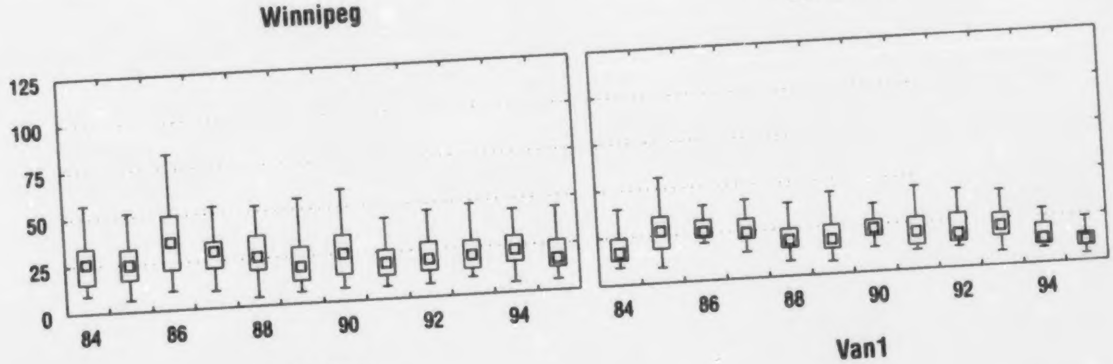
Egbert

Hamilton



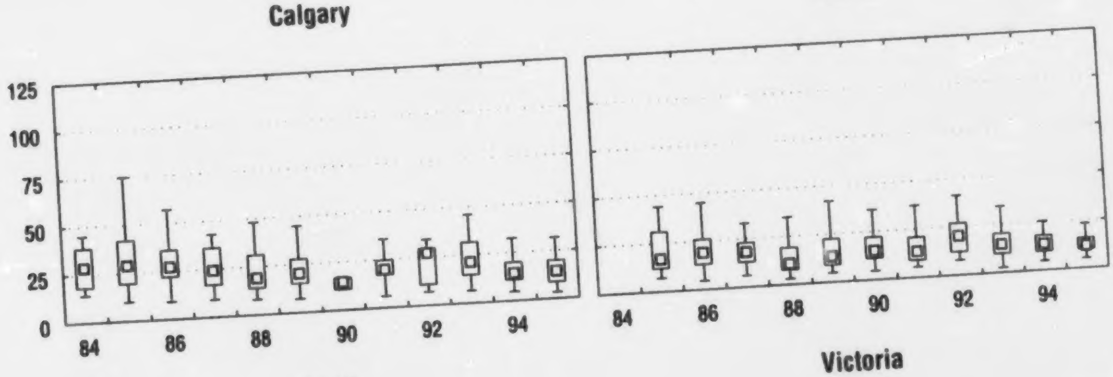
Edmonton

Winnipeg



Van1

Calgary



Victoria

Van2

Table 5.11 The Results of a Chi-Squared (χ^2) Test Applied to PM_{10} from Selected Dichotomous Sampler Sites

Station number and location		Data grouped by year		Data grouped by week day (weekend v mid-week)
			years used	
010101	Saint John	insufficient data		sign. at 95%
030115	Halifax	sign. at 99%	1985-1989, 1993	not sign.
030501	Kejimkujic	sign. at 99%	1984-1993	not sign.
05104	Montréal	insufficient data		sign. at 99%
05109	Montréal	sign. at 95%	1985-1989, 1993	not sign.
050307	Québec City	not sign.	1984-1992	not sign.
054101	Sutton	insufficient data		sign. at 90%
060104	Ottawa	sign. at 95%	1990-1993	not sign.
060204	Windsor	not sign.	1984-1989	not sign.
060211	Windsor	insufficient data		not sign.
060417	Toronto	sign. at 99%	1988-1993	not sign.
060424	Toronto	insufficient data		not sign.
061901	Walpole Is.	sign. at 95%	1984-1993	sign. at 99%
064401	Egbert	insufficient data		sign. at 99%
070119	Winnipeg	insufficient data		not sign.
090130	Edmonton	sign. at 90%	1987-1993	sign. at 99%
090204	Calgary	insufficient data		sign. at 95%
090227	Calgary	sign. at 99%	1985-1991	sign. at 95%
100106	Vancouver	sign. at 99%	1987-1989,1993	sign. at 99%
100111	Vancouver	not sign.	1985-1993	not sign.

Notes:

(1) only station 70119 in Winnipeg had sufficient data to calculate the χ^2 statistic by month. The result was significant at the 95% confidence level.

(2) The following categories of PM_{10} concentrations were used to group the observations of PM_{10} concentrations: 0-20, 21-40, 41-60, >60.

(3) χ^2 statistics were calculated only if all periods (i.e., every year, month and day) had >20 observations.

(4) Mid-week is defined here as Wednesday and Thursday.

The results of the test for differences between the PM_{10} concentration on weekends and mid-week (Wednesday and Thursday) were less uniform. One might expect sites in or near large cities with significant industrial activity to exhibit clear hebdomadal cycles of PM_{10} concentrations owing to the influence of the workweek upon emissions. This pattern was observed somewhat in the data from the NAPS sites, with most of the remote rural and urban non-industrial locations showing "not significant" in Table 5.11, indicating the absence of a significant day of week cycle. However, data from some sites located within major industrial centres also fail to exhibit a statistically significant day of week cycle according to this statistical test. Box plots of day of week variations in PM_{10} for selected sites were provided in Figure 5.6.

To further examine variability of PM_{10} concentrations on these time scales, the upper quartile mean (UQM) concentrations of the PM_{10} data were calculated. The statistics of PM_{10} records are dominated by a large number of low concentrations (see Figure 5.4), which can "mask" trends, or sample differences that are apparent only in the higher concentrations. Cleveland and McRae (1978) proposed a method for examining day-of-the-week variations of ozone concentrations that can be readily applied to PM_{10} data sets and on other time scales. They suggested calculating a "robust upper quartile mean of the daily maxima for each day of the week." The robust UQM of a data set is approximately equal to the average of the highest 25% of the observations and was calculated for the PM_{10} data sets using the following methodology, taken from Cleveland and McRae (1978):

1) The square root of each of the PM_{10} concentrations at a given site on a given year/month/day was calculated (denoted by X_i, i=1-n, where n is the number of days of observations). The square root of the PM_{10} concentrations is approximately normally distributed.
2) The median (m) of each X_i for each period and site was calculated.
3) The median of abs(X_i-m), was estimated for each time period and site. If any value of X_i-m is >3.5s, then that value was replaced by m+3.5s for positive values of X_i-m and m-3.5s for negative values of X_i-m, so that extreme values do not bias the analysis.
4) An average of the values above the 75th percentile of the X_i was calculated, and the result squared to return a value in mg/m^3. This is the robust mean of the upper quarter of the PM_{10} data.

In this way, a robust mean was calculated for each day of the week, month and year for data from each site. This value was then standardized by subtracting the average of the UQM calculated without stratification by day of the week, month or year and then dividing by this number. (This standardization facilitates comparison between sites that have different robust mean PM_{10} concentrations.) Each standardized UQM of the PM_{10} concentration shown in Figures 5.8 a, b and c represents a robust mean of about 10 values for each year, five for each month and day of the week. The results portrayed in Figure 5.8 confirm the findings of the χ^2 analysis. The UQM PM_{10} concentrations are typically, but not uniformly, lower on the weekends. Seasonality of the PM_{10} concentrations is evident, but the form of the seasonality is regionally variable. Inter-annual variability of the UQM PM_{10} concentrations is also evident, but it is difficult to determine any regional homogeneity at this time scale.

British Columbia

Thirteen monitoring locations that had more than 150 observations of 24 h average PM_{10} concentrations were selected to be discussed here (for locations see Table 5.4). Figure 5.9 summarizes the data from six of these sites by showing the interquartile range of PM_{10} concentrations by year, month and day of the week. Table 5.12 shows the frequency distribution of the PM_{10} data from all 13 sites. The range of the extreme PM_{10} concentrations is similar to that from the NAPS sites. Table 5.13 shows the results of χ^2 statistics as applied to PM_{10} concentrations conditionally sampled by year, month and weekday. From Figure 5.9 and Table 5.13, it is clear that several of the sites show variability of PM_{10} concentrations on annual, seasonal and hebdomadal (weekly) time scales.

Figure 5.8 Standardized Upper Quartile Mean (UQM) PM_{10} Concentrations at National Network Sites by a) Year b) Month and c) Day of Week.

(a)

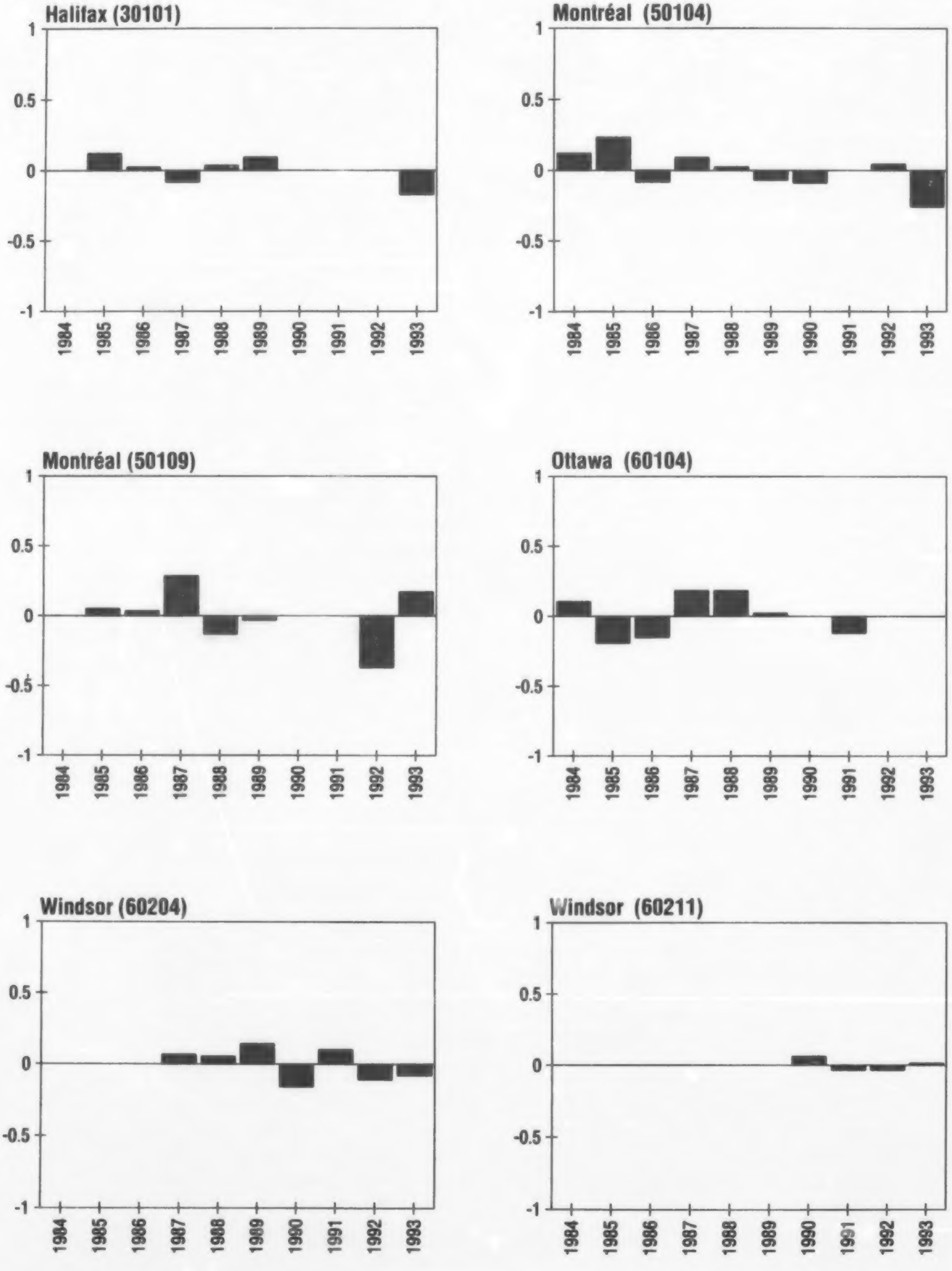


Figure 5.8 Standardized Upper Quartile Mean (UQM) PM_{10} Concentrations at National Network Sites by a) Year b) Month and c) Day of Week. *(continued)*

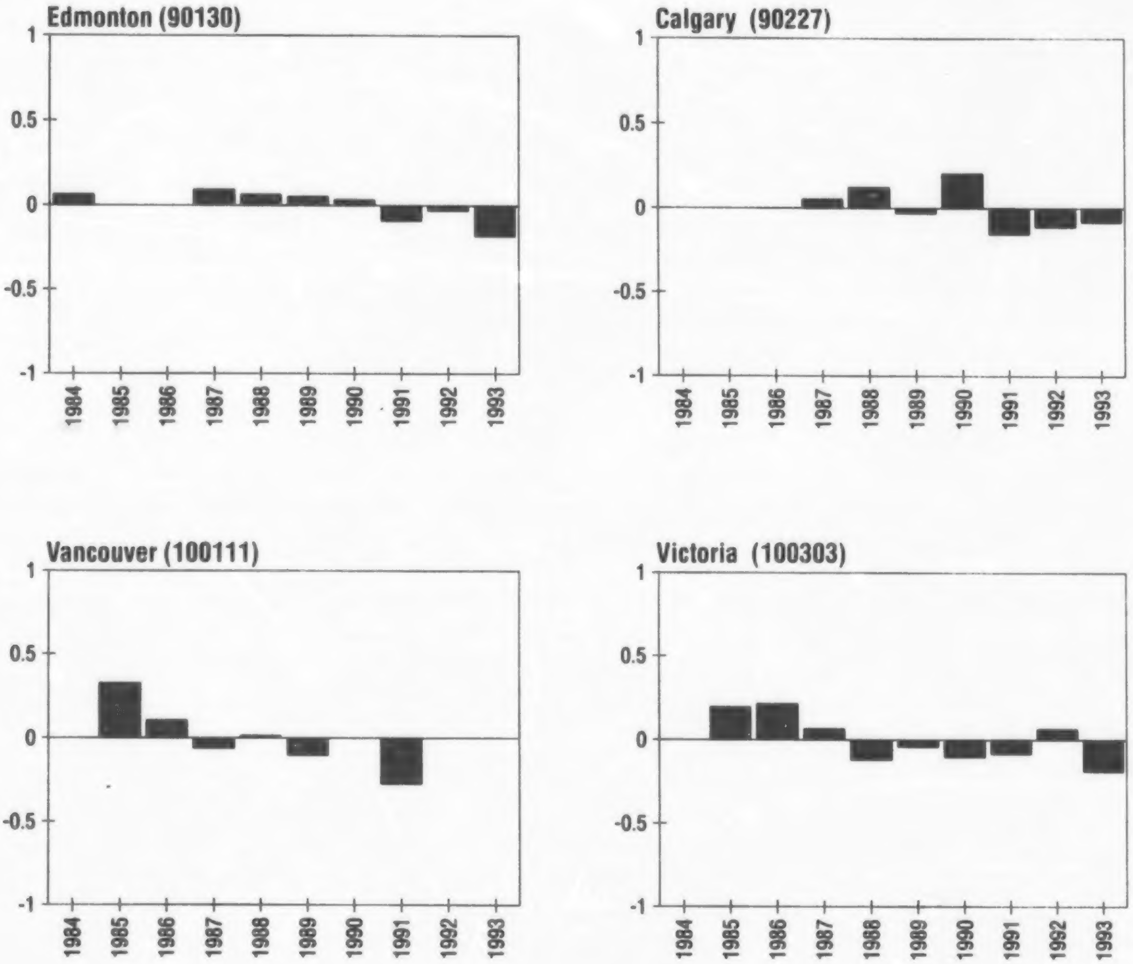
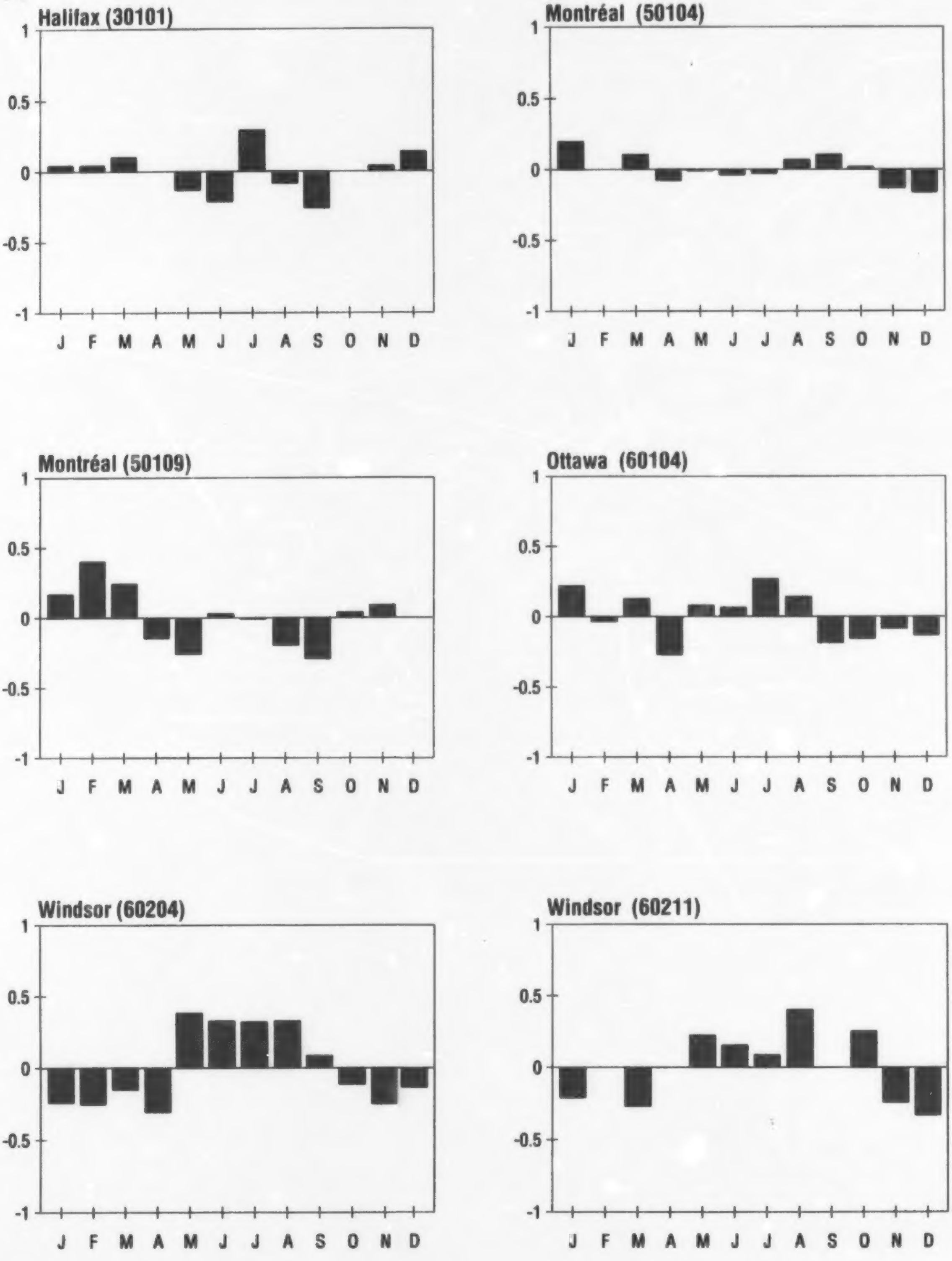


Figure 5.8 Standardized Upper Quartile Mean (UQM) PM_{10} Concentrations at National Network Sites by a) Year b) Month and c) Day of Week. ***(continued)***

(b)



Halifax (30101)

Montréal (50104)

Montréal (50109)

Ottawa (60104)

Windsor (60204)

Windsor (60211)

Figure 5.8 Standardized Upper Quartile Mean (UQM) PM_{10} Concentrations at National Network Sites by a) Year b) Month and c) Day of Week. *(continued)*

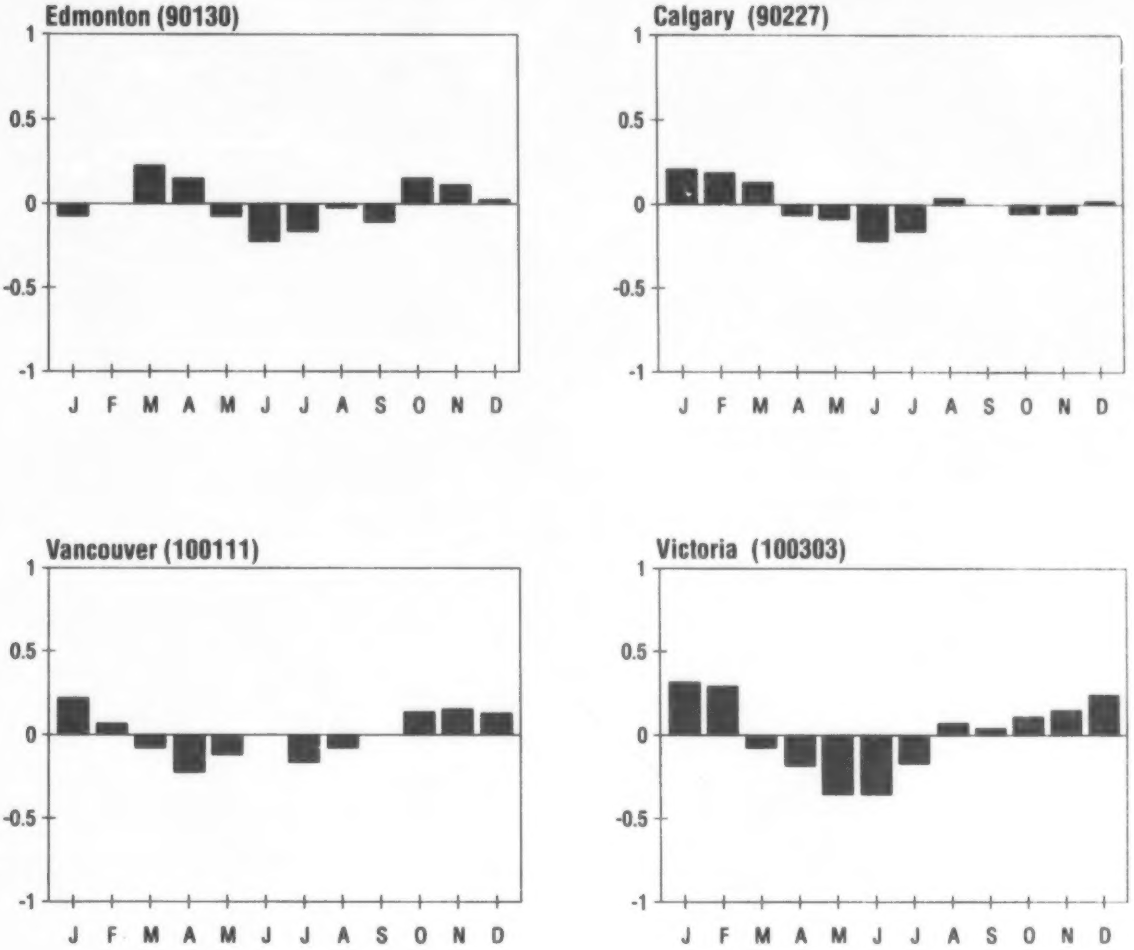


Figure 5.8 Standardized Upper Quartile Mean (UQM) PM_{10} Concentrations at National Network Sites by a) Year b) Month and c) Day of Week. ***(continued)***

(c)

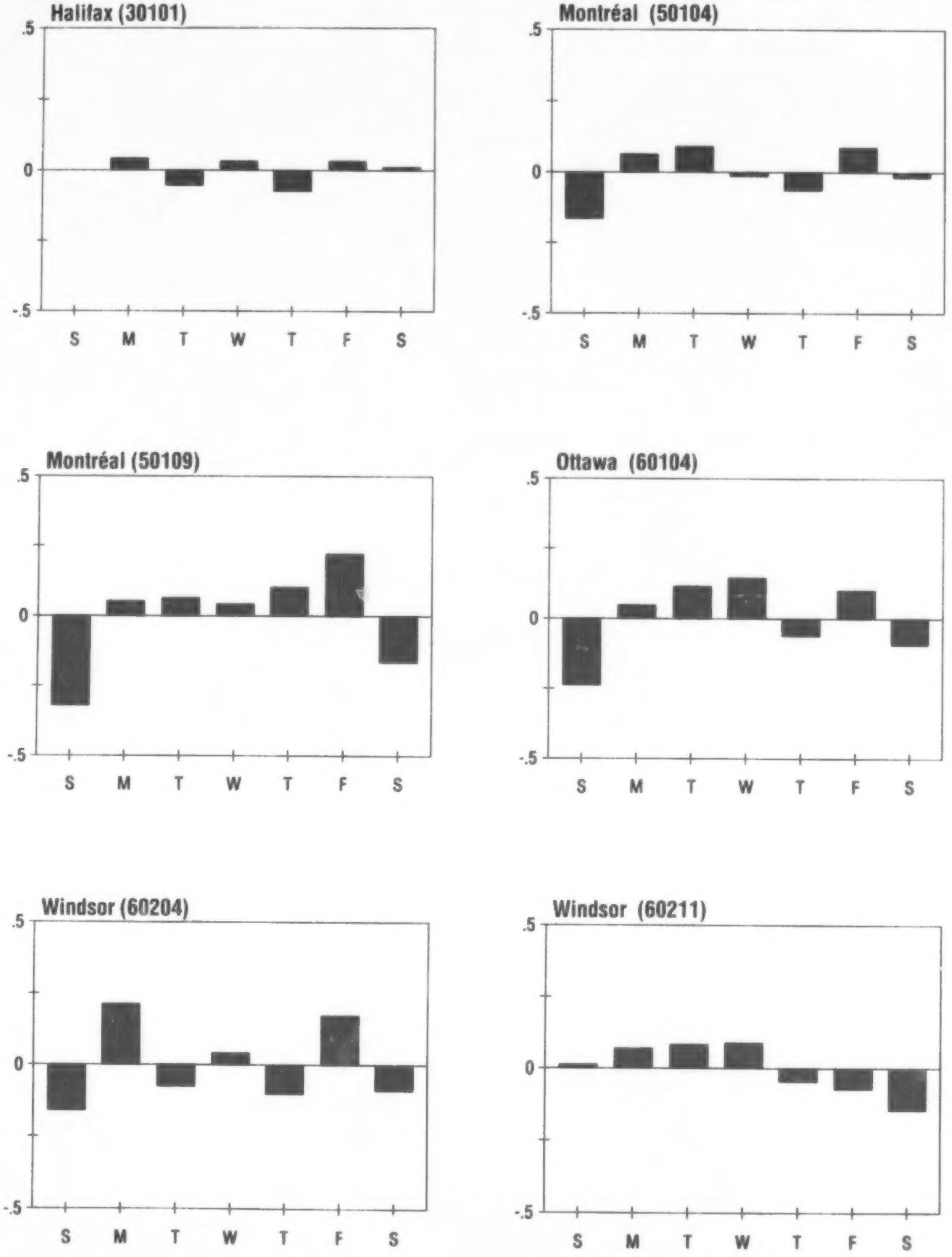


Figure 5.8 Standardized Upper Quartile Mean (UQM) PM_{10} Concentrations at National Network Sites by a) Year b) Month and c) Day of Week. *(continued)*

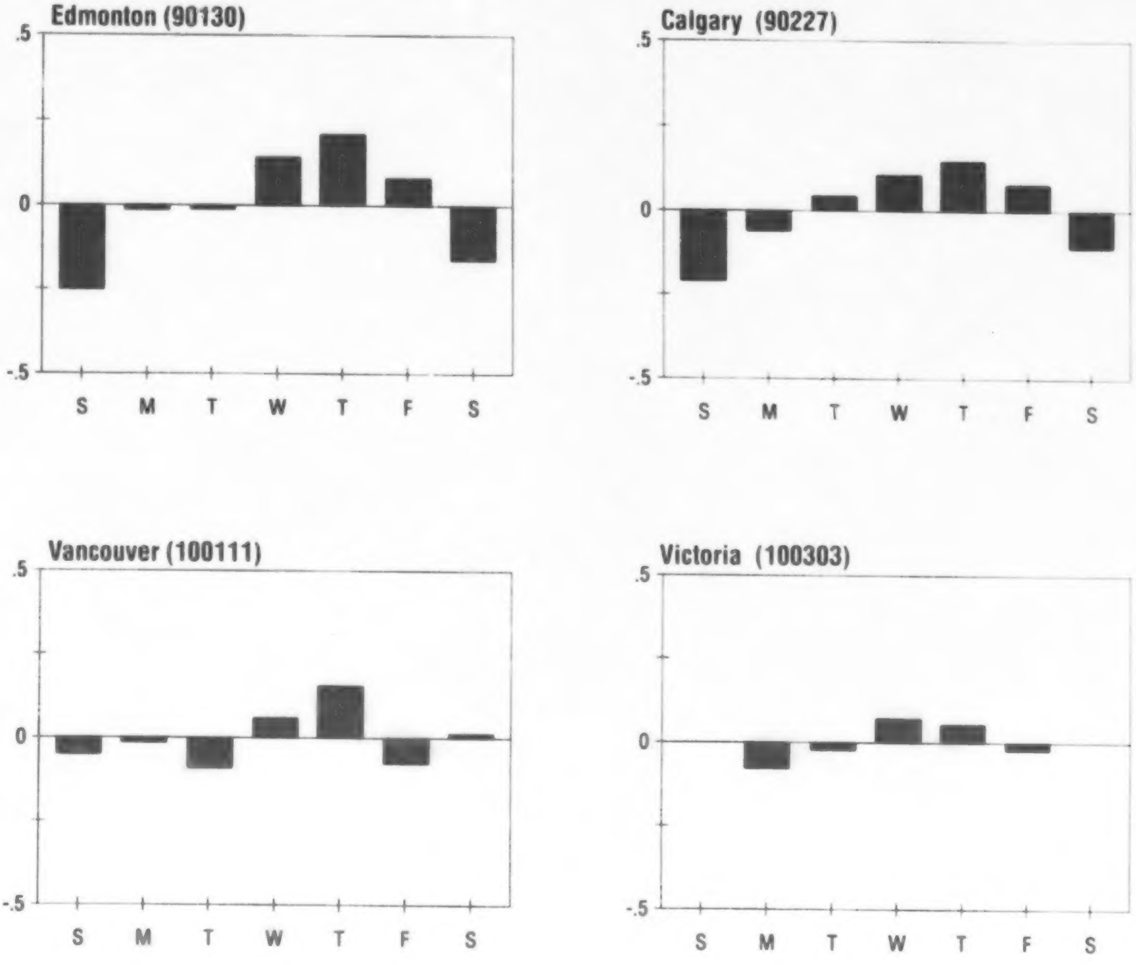


Figure 5.9 The Interquartile Range of PM_{10} Concentrations (µg/m³) from Monitoring Sites in BC by Year, Month and Day of Week.

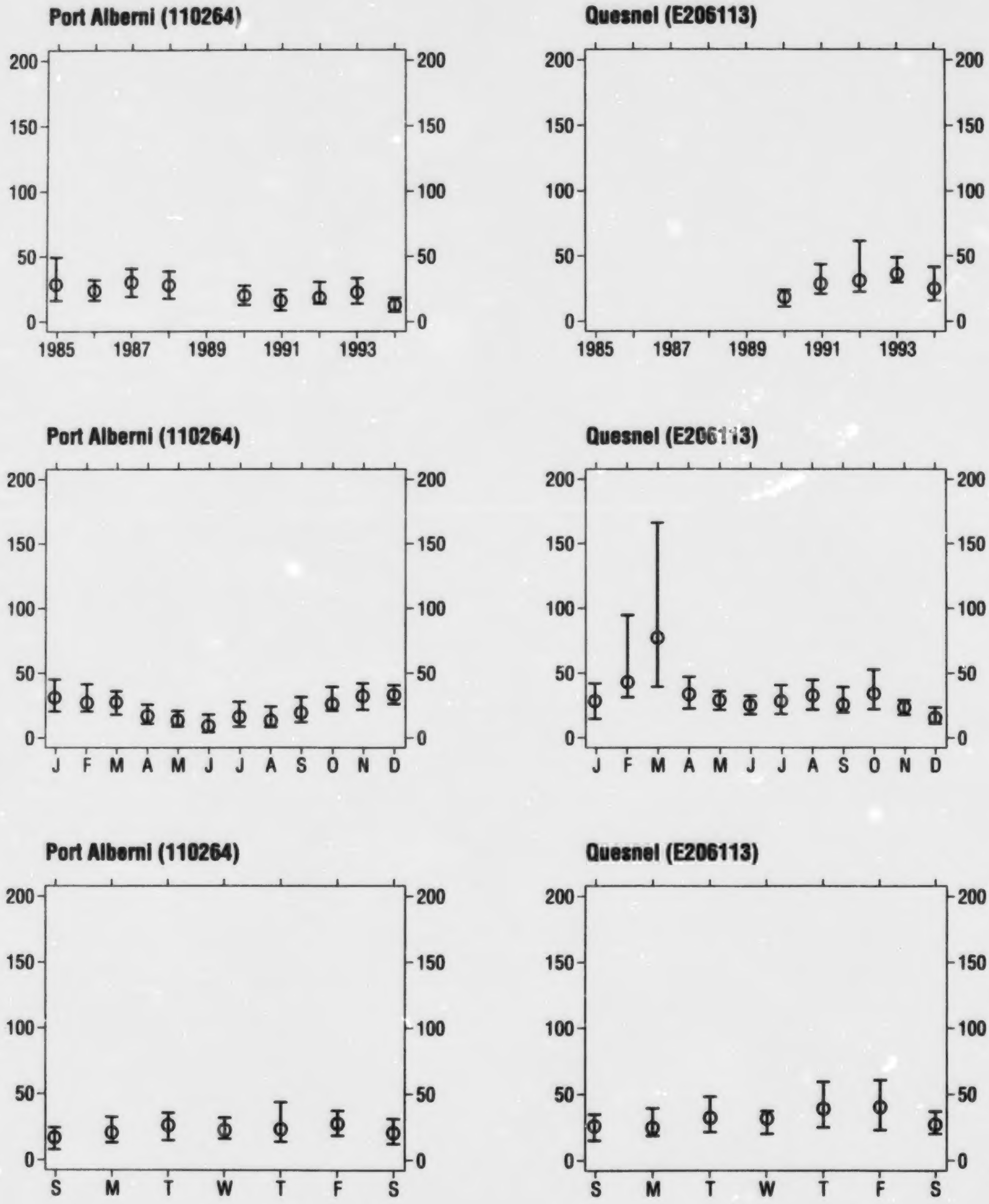


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Figure 5.9 The Interquartile Range of PM_{10} Concentrations (μg/m³) from Monitoring Sites in BC by Year, Month and Day of Week. *(continued)*

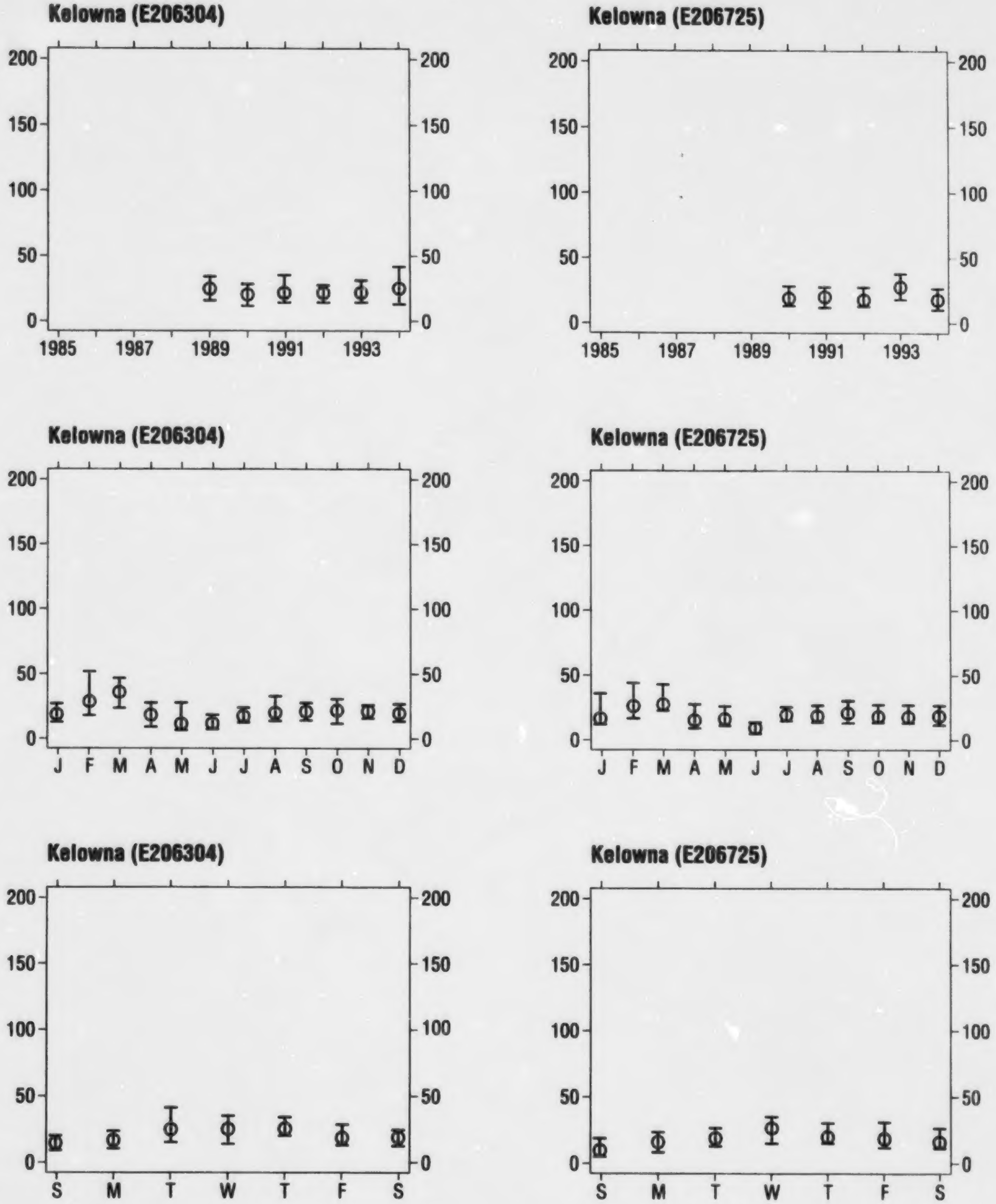


Figure 5.9 The Interquartile Range of PM_{10} Concentrations (µg/m³) from Monitoring Sites in BC by Year, Month and Day of Week. *(continued)*

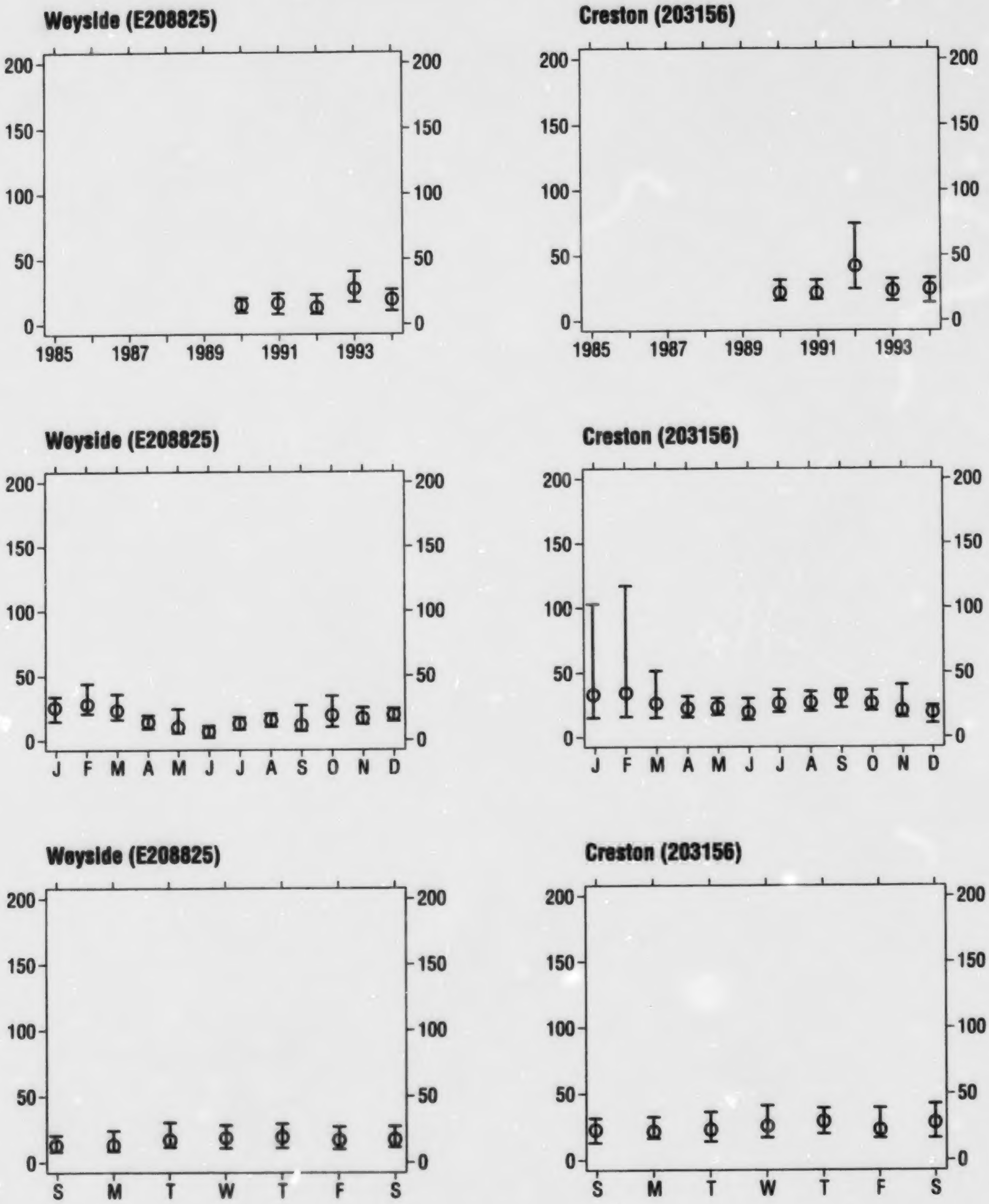


Table 5.12 Frequency Distributions of 24 h Average PM_{10} Concentrations ($\mu g/m^3$) for Selected Sites in British Columbia.

Station number and location		Arith. Mean	Min	Concentration at the nth percentile					Max
				10	30	50	70	90	
110264	Port Alberni	25.1	2	8	14	21	30	48	100
605002	Kamloops	20.5	3	7	13	17	23	37	86
E206112	Williams Lake	30.9	3	11	18	24	35	60	125
E206113	Quesnel	40.8	4	12	22	31	39	70	391
E206169	Pitt Meadows	16.8	2	5	9	15	21	32	47
E206241	Cranbrook	44.4	7	12	21	35	57	89	196
E206304	Kelowna	25.7	2	10	16	22	28	46	130
E206589	Smithers	22.6	2	7	13	18	27	41	169
E206725	Kamloops	25.4	2	11	16	22	29	43	95
E206931	Castlegar	27.0	2	12	18	24	30	46	78
E208805	Weyside	22.9	2	9	14	19	26	43	95
E213056	Creston	35.0	2	11	20	25	35	64	419
E213114	Chilliwack	19.0	2	7	12	17	22	33	108

The following inferences may be drawn based upon the results of the χ^2 statistics and Figure 5.9:

1) The year-to-year variation of PM_{10} concentrations is significant at over half of the stations for which sufficient data are available to test the distributions. Note that this is not necessarily indicative of a significant trend in the data. As shown in Figure 5.9, the interquartile range of PM_{10} concentrations at these sites shows no evidence of a trend. Inter-annual variability is often noted in other air quality parameters and may reflect the influence of both changing emission patterns/amounts and the inter-annual variability of meteorological conditions (e.g., Pryor et al., 1995c). The inter-annual variability of both PM_{10} concentrations *and* prevailing meteorology indicates the value of having long-term monitoring stations for use in trend analysis. It also emphasizes the importance of declimatizing air quality data before attempting trend analysis if the implication of a "trend" (positive or negative) is going to be interpreted as evidence of changing emission practices (Pryor et al., 1995c).

2) For most sites, there were insufficient data to examine month-to-month or seasonal variability of PM_{10} concentrations. However, the inference may be drawn from Figure 5.9 that there is seasonality of PM_{10} concentrations. For example, qualitatively, it appears that Quesnel (E206113), Kelowna (E206304) and Kamloops (E206725) exhibit elevated PM_{10} concentrations in March. The winter maximum of PM_{10} concentrations at Port Alberni (110264) is a recurrent feature in the data on an inter-annual basis and may reflect the influence of increased wood burning or decreased mixing depth in the winter. Only data from 110264 (Port Alberni) could be tested using the χ^2 statistic, and it produced a statistically significant result.

3) The χ^2 statistic for mid-week-weekend differences in PM_{10} concentrations is significant at eight of the 13 sites. As shown in Figure 5.9, PM_{10} concentrations are typically highest during the mid-week period and lowest on the weekends. This data feature is also apparent in the NAPS data (see Figure 5.6) and has been observed in data from the Los Angeles Basin (e.g., Hoggan et al., 1989).

Table 5.13 The Results of a Chi-Squared (χ^2) Test Applied to PM_{10} Data from British Columbia.

Station number and location		**Data grouped by year**		**Data grouped by week day (weekend v mid week)**
			years used	
110264	Port Alberni	sign. at 99%	1985-1994 (1989 missing)	sign. at 99%
605002	Kamloops	sign. at 90%	1990-1993	sign. at 95%
E206112	Williams Lake	not sign.	1991-1994	sign. at 99%
E206113	Quesnel	sign. at 95%	1991-1994	not sign.
E206169	Pitt Meadows	sign. at 99%	1991-1994	sign. at 95%
E206241	Cranbrook	insufficient data		not sign.
E206304	Kelowna	not sign.	1989-1993	sign. at 99%
E206589	Smithers	not sign.	1990-1993	not sign.
E206725	Kamloops	sign. at 90%	1990-1994	sign. at 99%
E206931	Castlegar	insufficient data		not sign.
E208805	Weyside	sign. at 99%	1990-1994	sign. at 90%
E213056	Creston	sign. at 99%	1991-1994	not sign.
E213114	Chilliwack	not sign.	1991-1994	sign. at 99%

Notes:

(1) Only station 110264 had sufficient data to calculate the χ^2 statistic by month. The result was significant at the 99% confidence level.

(2) χ^2 statistics were calculated only if all time periods (i.e., every year, month and day of the week) had >20 observations.

(3) Mid-week is defined here as Wednesday and Thursday.

The UQM PM_{10} concentrations shown in Figure 5.10 are calculated by year, month and day of the week for the NAPS data. The results of this calculation are in accord with the findings of the χ^2 analysis. Weekday PM_{10} concentrations are typically, but not uniformly, higher than those recorded during the weekends. PM_{10} concentrations are typically highest during the winter (January-March) and show considerable inter-annual variability. Although median PM_{10} concentrations from 1990 are not atypical, 1990 UQM PM_{10} concentrations are lower than during other years. It may be inferred that 1990 was characterized by a lower-than-average frequency or intensity of high particulate loading episodes. Conversely, UQM PM_{10} concentrations from 1993 at five of the six sites shown in Figure 5.10 exhibit above-average standardized values.

Figure 5.10 Standardized Upper Quartile Mean (UQM) PM_{10} Concentrations at British Columbia Sites by a) Year b) Month and c) Day of Week.

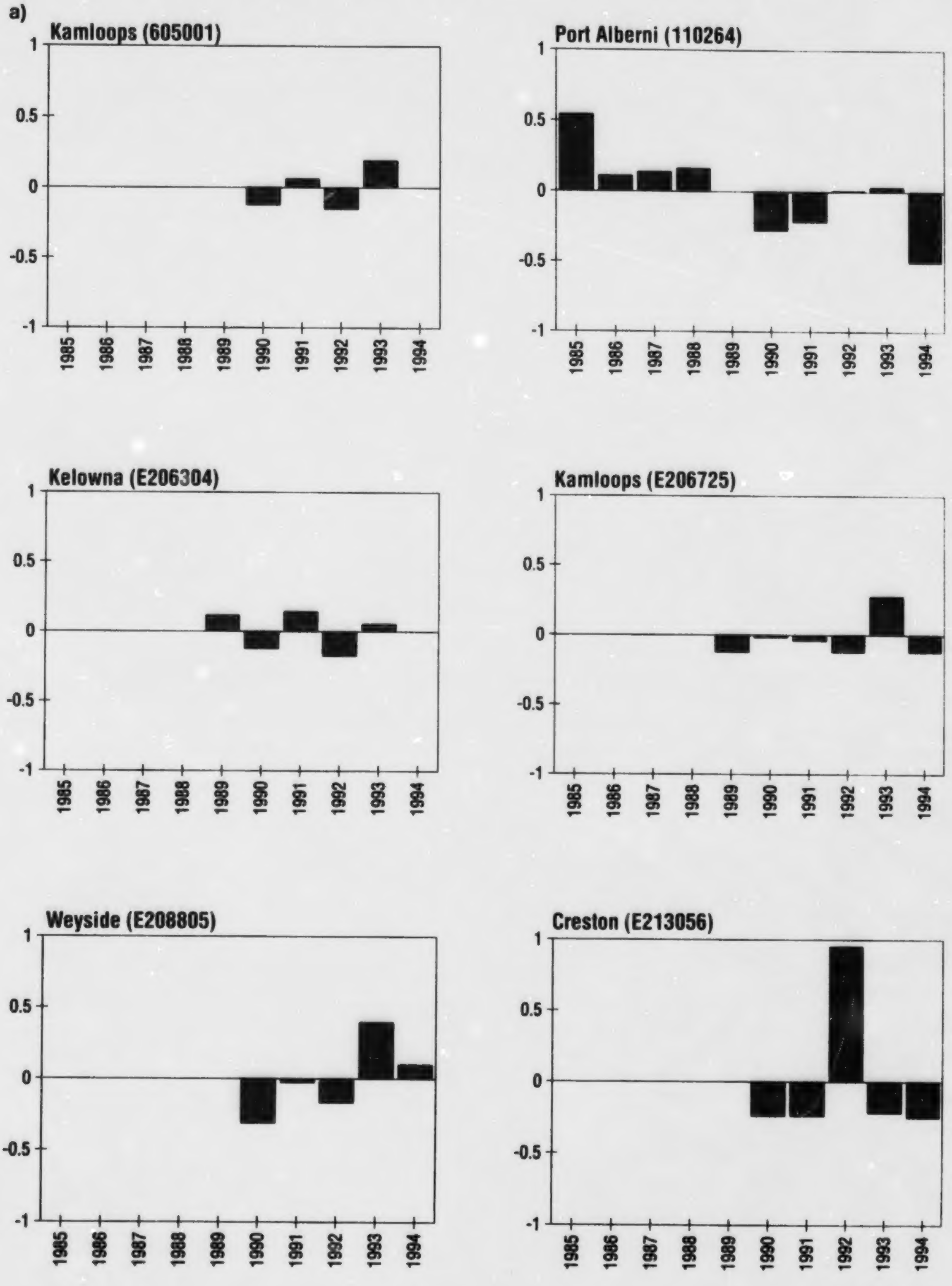


Figure 5.10 Standardized Upper Quartile Mean (UQM) PM_{10} Concentrations at British Columbia Sites by a) Year b) Month and c) Day of Week. *(continued)*

b)

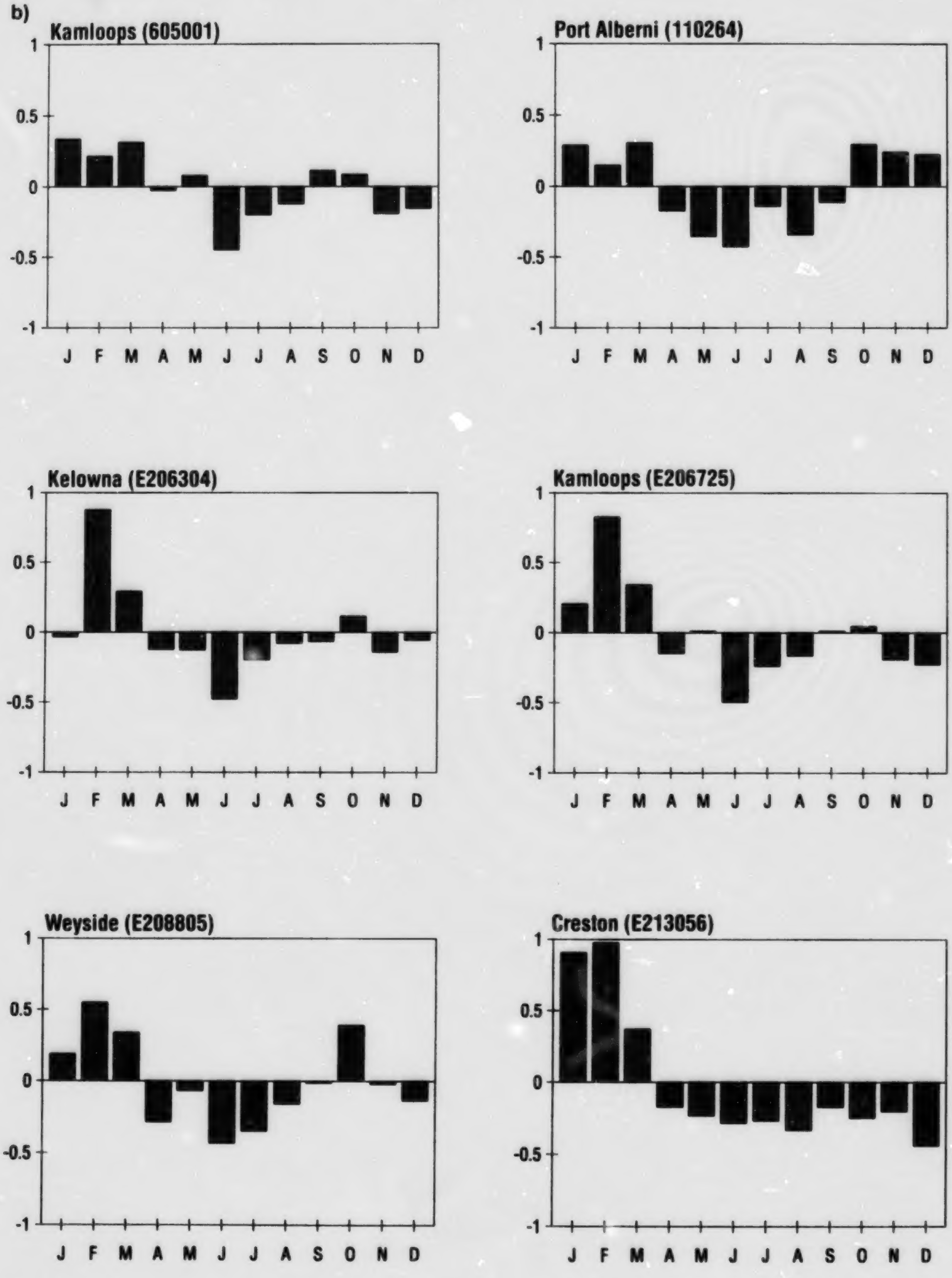


Figure 5.10 Standardized Upper Quartile Mean (UQM) PM_{10} Concentrations at British Columbia Sites by a) Year b) Month and c) Day of Week. *(continued)*

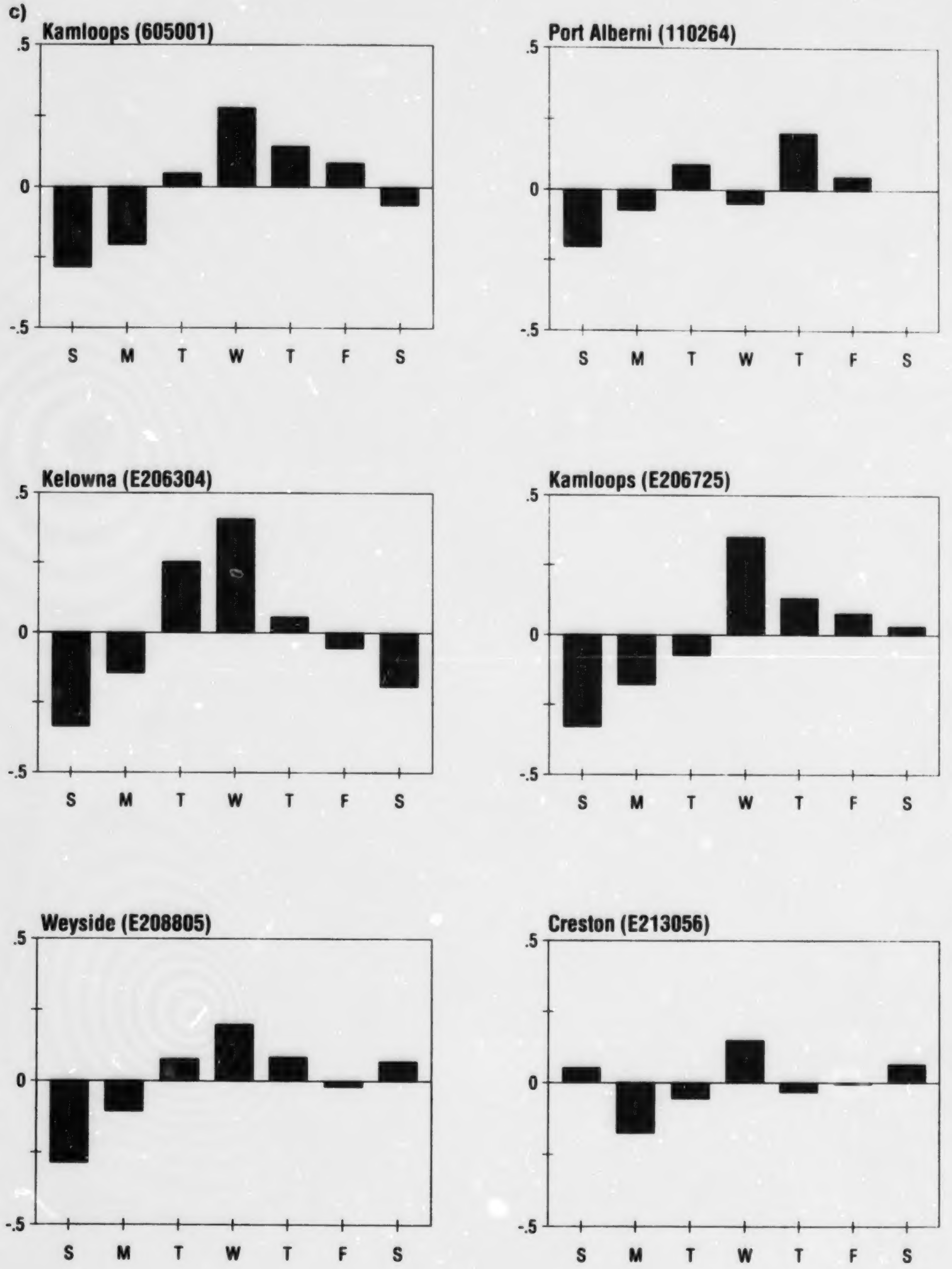


Figure 5.11 The Interquartile Range of PM_{10} Concentrations (µg/m³) from Monitoring Sites in Ontario by Month and Day of Week.

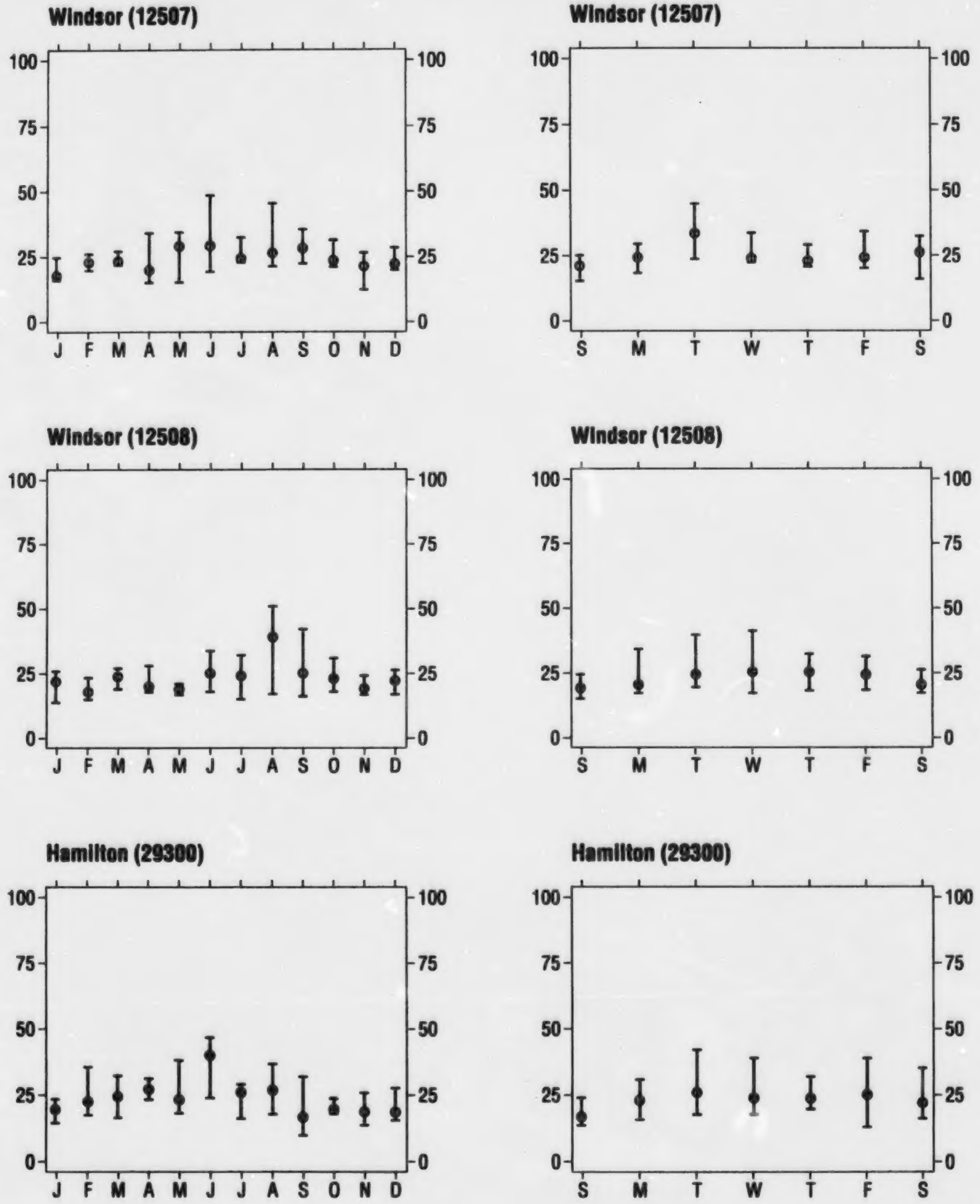


Figure 5.11 The Interquartile Range of PM_{10} Concentrations (µg/m³) from Monitoring Sites in Ontario by Month and Day of Week. *(continued)*

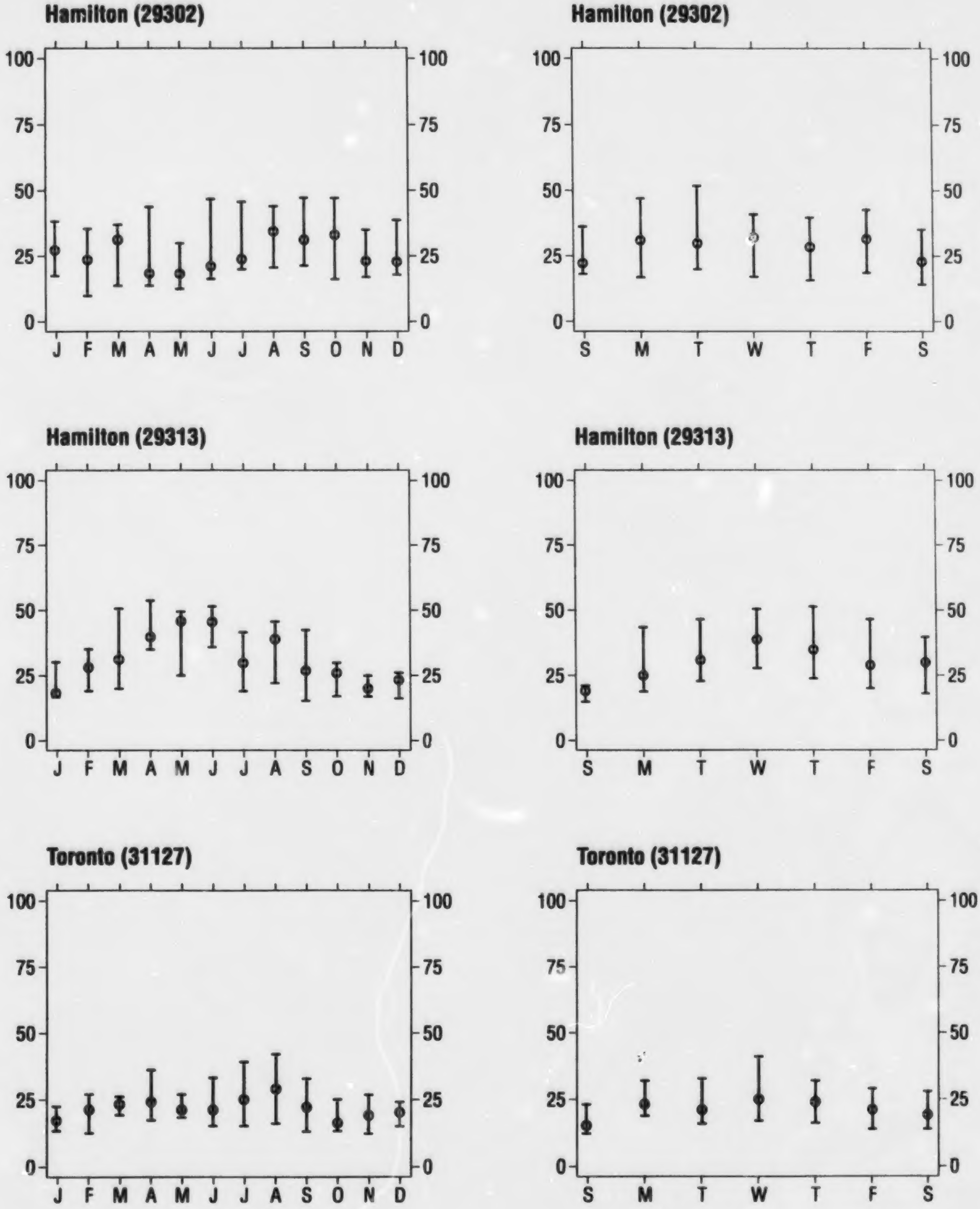
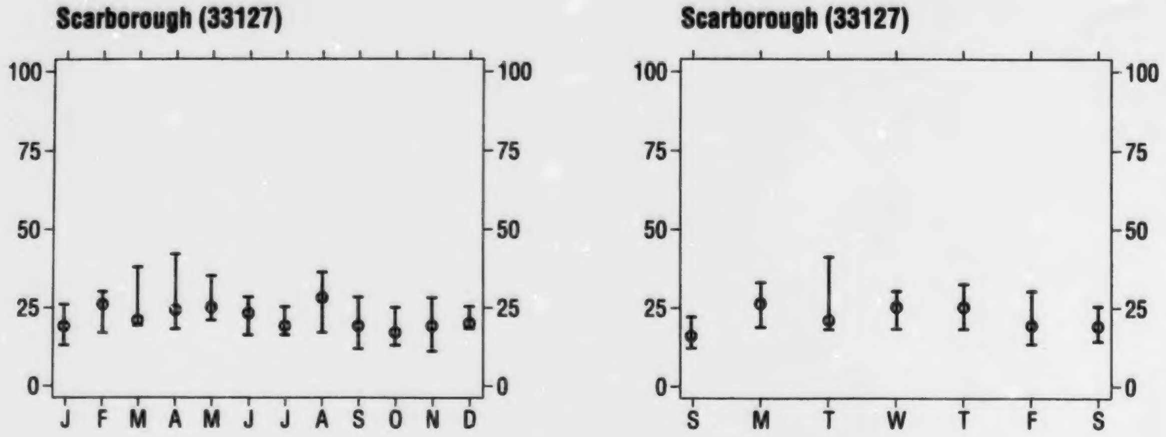
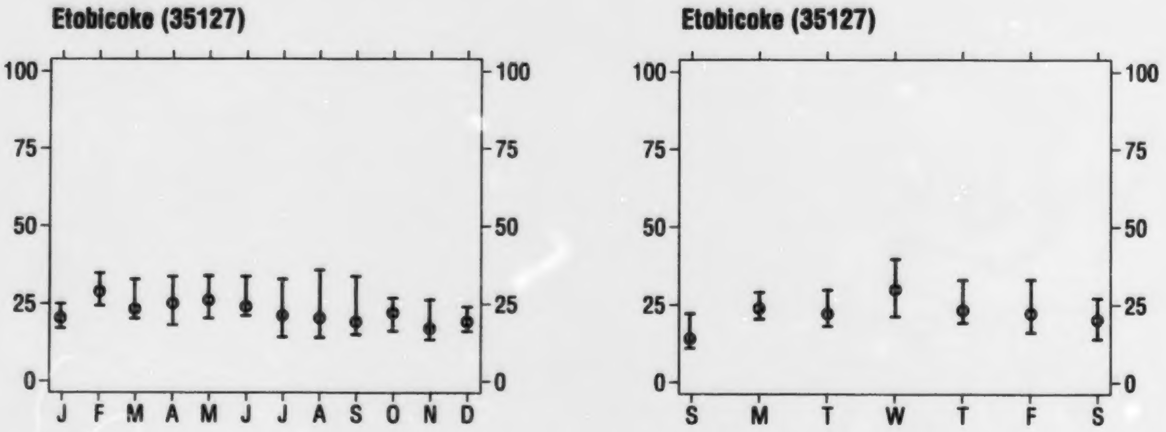


Figure 5.11 **The Interquartile Range of PM_{10} Concentrations (µg/m³) from Monitoring Sites in Ontario by Month and Day of Week.** ***(continued)***





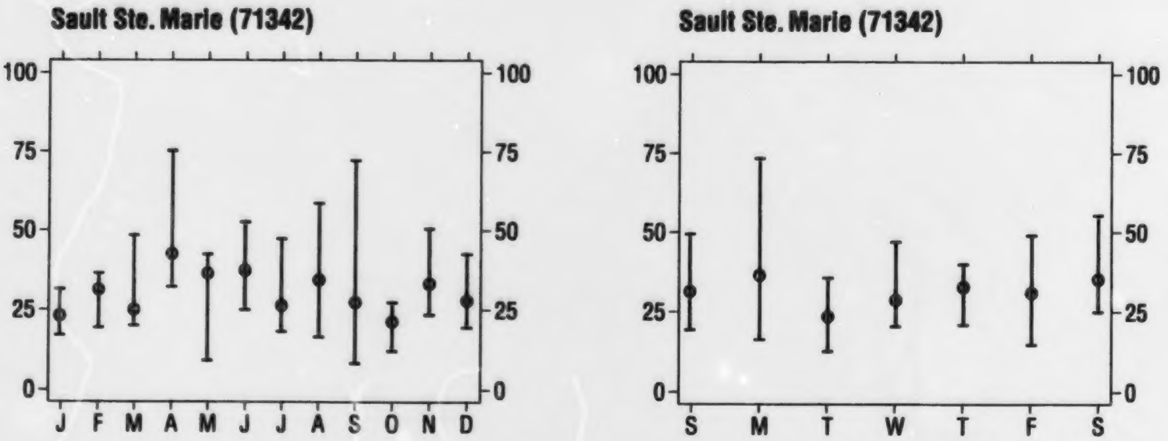
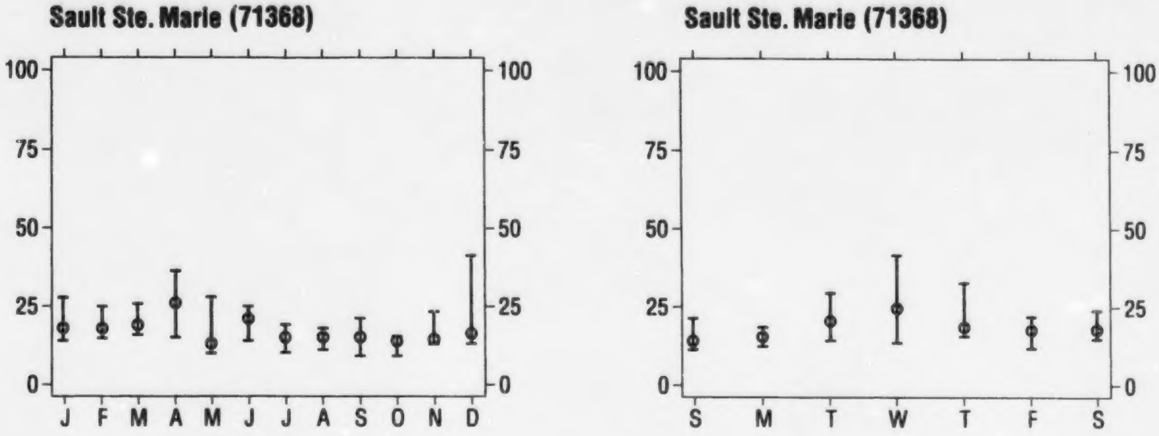


Figure 5.11 The Interquartile Range of PM_{10} Concentrations (µg/m³) from Monitoring Sites in Ontario by Month and Day of Week. ***(continued)***



Ontario

Table 5.14 summarizes the frequency distributions of PM_{10} concentration data from 18 of the PM_{10} network sites currently operated as part of the Ontario Inhalable Particulates Network. Again, the frequency distributions are similar to those that characterize the NAPS and BC sites. Figure 5.11 shows the interquartile ranges of these data by month and day of the week. Unfortunately, the data were not available to calculate the χ^2 statistics or UQM concentrations. It is interesting to note however, that the data typically exhibit a summertime maximum, as observed in the NAPS data from Ottawa and Windsor, and a weak mid-week maximum.

Table 5.14 Frequency Distributions of 24 h Average PM_{10} Concentrations (µg/m³) for Sites in Ontario.

Site #		Year		Mean		Min	Percentiles					Max
		From - To		Arith.	Geom		10	30	50	70	90	
12507	Windsor	90	93	27	25	9	14	21	25	30	48	118
12508	Windsor	89	93	27	24	10	14	18	23	30	47	84
15516	London	90	93	19	16	5	9	12	15	21	36	56
22304	Nanticoke	92	94	17	14	1	7	10	14	18	31	69
27308	St. Catherines	91	93	22	19	2	10	15	19	23	37	70
27352	Thorold	90	93	33	29	9	14	22	27	35	60	100
29300	Hamilton	90	93	28	24	6	13	18	23	32	49	97
29302	Hamilton	89	93	32	27	6	10	20	30	39	55	130
29313	Hamilton	90	93	35	30	8	15	21	30	42	59	110
29324	Hamilton	92	93	24	20	4	10	14	18	27	41	79
31127	Toronto	89	93	27	23	7	12	17	21	28	47	120
33127	Scarborough	90	93	26	22	3	11	18	21	28	44	100
35127	Etobicoke	90	93	26	23	9	13	19	22	30	41	81
44127	Oakville	92	93	22	19	4	8	15	19	25	36	92
63201	Thunder Bay	89	93	19	16	4	8	12	16	22	33	63
71342	Sault Ste. Marie	89	93	40	30	4	10	21	32	44	77	190
71368	Sault Ste. Marie	90	93	23	19	4	10	14	18	24	41	100
77326	Sudbury	91	93	18	16	5	9	12	14	20	32	59

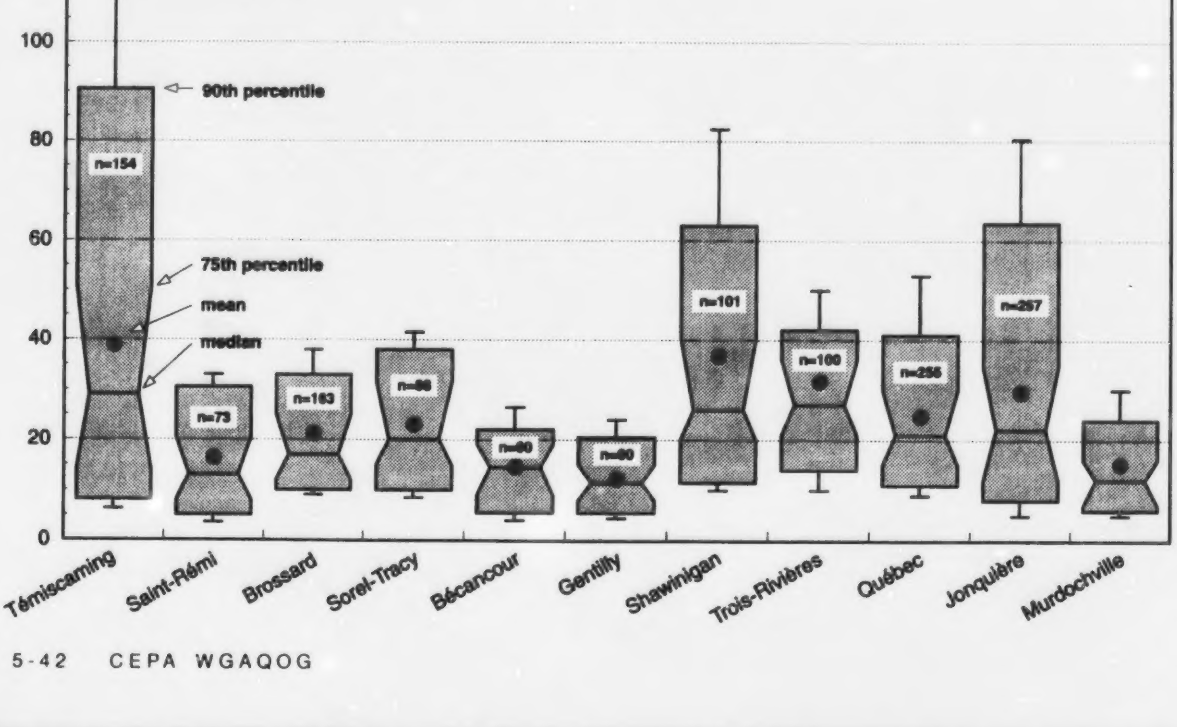
Québec

Results from 11 provincial monitoring sites are summarized in Table 5.15 and in Figure 5.12. A number of these sites are impacted by industrial sources and show generally higher PM_{10} maxima and 90th percentiles than nonindustrial urban locations. The sites in Témiscaming, Shawinigan, Trois-Rivières and Jonquière recorded the highest values with mean PM_{10} concentrations ranging from 29 to 39 µg/m³ and 24 h maxima ranging from 70 to 323 µg/m³. At the Témiscaming site, 25% of the readings were greater than or equal to 50 µg/m³. An analysis of seasonal and day of week variability was not carried out for these sites.

Table 5.15 Frequency Distributions of 24 h Average PM_{10} Concentrations (µg/m³) for Sites in Québec.

Prov. ID	City	N	Percentiles					Max.	Mean	Geometric Mean
			10	25	50	75	90			
08401	Témiscaming	154	10	14	29	50	86	210	39	27
06802	Saint-Rémi	73	7	8	13	20	30	52	15	13
06760	Brossard	163	11	12	18	25	33	69	20	18
06672	Sorel-Tracy	88	11	15	20	31	37	65	23	20
04505	Bécancour	60	7	9	15	19	22	33	14	12
04504	Gentilly	60	7	8	12	15	20	33	12	11
04130	Shawinigan	101	14	20	26	40	62	323	35	27
04019	Trois-Rivières	100	16	21	27	36	41	70	29	26
03006	Québec	255	12	15	21	31	41	70	24	20
02016	Jonquière	257	10	12	22	36	63	280	29	24
01804	Murdochville	101	7	7	12	16	24	59	14	12

Figure 5.12 Distribution of PM_{10} Concentrations (µg/m³) at Quebec Monitoring Sites 1991-1995.



Summary

Mean 24 h PM_{10} concentrations in Canadian cities are between 4 and 8 times the background levels estimated by Trijonis et al. (1990) for the western and eastern United States (Table 5.2). The clear implication is that anthropogenic activities make a substantial contribution to ambient PM_{10} levels.

Although the mean 24 h PM_{10} concentrations recorded at monitoring sites throughout Canada fall within a relatively narrow range, there is significant variability of measured PM_{10} concentrations on both temporal and spatial scales. The highest PM_{10} concentrations recorded by the NAPS monitoring network were observed in Québec and Ontario (at sites in Montréal, Windsor, Toronto and Walpole Island) and at a single site in Calgary, Alberta. The Windsor-Québec City corridor has previously been identified as a region of poor air quality (McKendry, 1993). However, even within this area, some sites experience comparatively low ambient PM_{10} levels when the entire distribution of PM_{10} measurements is examined (e.g., Ottawa and Québec City). Comparison of measurements from national and provincial networks indicates that the extremes of the frequency distribution of PM_{10} measurements at sites within the same province and even within the same city show a high degree of variability. Local sources of particulate emissions can play a significant role in determining ambient PM_{10} concentrations.

PM_{10} concentrations also vary by month, day of the week and hour of the day. The season of maximum PM_{10} concentrations appears to be regionally variable, reflecting variations in dominant sources of PM_{10} (especially secondary aerosols) and synoptic meteorology. In accord with Figure 5.5 and 5.8b, the sites that exhibit the highest degree of seasonality in the upper quartile of PM_{10} concentrations are in Windsor (a summertime maximum) and Victoria (a wintertime maximum). Many of the sites in British Columbia seem to exhibit late winter-spring maxima of both mean and median PM_{10} concentrations and the upper quartile of the distribution (Figure 5.10). This indicates that both average and extreme PM_{10} concentrations are typically higher during the months of January, February and March. Sites in the Ontario Inhalable Particulates Network seem to exhibit a summertime maximum of the interquartile range and monthly mean PM_{10} concentrations (in accord with Ontario data from the NAPS network). These provincial differences may reflect the greater abundance of secondary aerosols in the Windsor-Québec City corridor, where precursor concentrations are known to be high. A hebdomadal cycle of PM_{10} concentrations is evident at most sites described here. Typically, weekend concentrations of PM_{10} are lower than those observed during the workweek. The sites that do not exhibit this cycle tend to be located in rural environments.

PM_{10} data from three networks operated within Canada have been presented in the form of frequency distributions, average concentrations and UQMs, so that the entire distribution of PM_{10} concentrations may be examined. However, it should be noted that the current strategy of monitoring one day in six does not permit the extremes of the concentration distribution to be accurately quantified. The one-in-six-day schedule likely underestimates the frequency of PM_{10} extreme events because the nearest days to the event day, and on the event day itself, may be missed by the sampling schedule. The values for the lowest percentiles (30th percentile and below) reflect the lowest measured concentrations and so are impacted by the minimum detectable limit (MDL), treatment of values below the MDL and instrument/measurement accuracy. These may vary by instrument and collection agency.

5.3.4 24 h Average $PM_{2.5}$ Concentrations

National Network (NAPS)

Frequency distributions of $PM_{2.5}$ concentrations for the dichotomous sampler sites (Table 5.3) are provided in Table 5.16. Mean concentrations at the urban sites ranged from 8.5 to 20.2 $\mu g/m^3$. The highest $PM_{2.5}$ concentrations (in terms of means and 90th percentiles) were measured at sites in Montréal, Windsor, Hamilton, Walpole Island, Toronto and Vancouver. Most of these same sites also recorded the highest PM_{10} concentrations. The three rural sites of Kejimkujik, Sutton and Egbert record mean $PM_{2.5}$ concentrations of 7.0, 7.7 and 10.5 $\mu g/m^3$ respectively, although observations were only available for 1992-1995 for Kejimkujik and Egbert and for May-September, 1993 for Sutton. As noted, relatively high $PM_{2.5}$ values were recorded at the other rural site at Walpole Island. This may be due to the effect of nearby agricultural operations and due to its proximity to metropolitan Detroit.

Table 5.16 Frequency Distributions of 24 h Average PM$_{2.5}$ Concentrations (μg/m^3) from Dichotomous Sampler Sites (1984-1995) – National Network.

NAPS ID	City	N	Min.	Percentiles					Max.	Mean	Std. Dev.
				10.0	30.0	50.0	70.0	90.0			
10101	St. John's	39	1.0	3.0	6.0	9.0	11.0	18.0	26.0	9.8	5.7
40203	Saint John	821	0.6	2.5	4.8	7.2	10.0	16.2	53.2	8.5	6.2
30101	Halifax	335	2.8	6.0	10.0	12.0	15.6	25.0	45.5	14.0	7.1
30118	Halifax	273	0.8	4.6	6.0	7.4	9.6	15.1	43.4	9.0	5.5
30119	Dartmouth	83	0.7	3.1	4.5	6.3	7.7	10.9	23.5	6.9	4.0
30501	Kejimkujik	506	0.4	2.0	3.3	4.8	7.3	13.6	46.7	7.0	7.1
50104	Montréal	849	1.4	5.3	8.9	12.5	17.8	29.0	69.6	15.2	10.3
50109	Montréal	373	0.6	7.4	12.4	17.0	23.0	36.0	89.0	20.2	13.0
50307	Québec City	267	0.4	4.0	6.9	9.0	13.0	23.0	49.0	11.5	8.1
50308	Québec City	40	0.1	3.0	4.5	7.3	9.1	19.1	32.9	9.0	7.2
54101	Sutton	136	1.0	2.4	4.1	6.1	9.1	15.6	33.2	7.7	5.5
60104	Ottawa	569	1.2	3.9	6.0	9.0	14.0	24.0	53.8	12.0	8.8
60204	Windsor	367	1.8	6.5	11.0	15.5	21.6	32.2	70.6	17.9	11.1
60211	Windsor	615	1.8	6.7	10.3	15.0	19.7	31.2	85.6	17.4	11.3
60417	Toronto	282	1.4	6.4	10.0	14.0	19.0	29.0	71.0	16.4	10.5
60424	Toronto	599	1.3	5.2	8.1	12.6	18.4	30.4	66.4	15.5	10.6
60403	Toronto	96	3.1	5.8	9.5	14.8	20.1	28.0	43.0	15.9	9.0
60512	Hamilton	329	1.9	5.6	10.4	15.2	22.2	33.4	74.1	18.1	12.1
61901	Walpole Island	330	2.0	4.4	8.7	13.3	21.0	36.6	126.6	17.9	14.8
64401	Egbert	304	0.7	2.6	4.8	7.7	12.6	21.9	47.7	10.5	8.5
70119	Winnipeg	567	0.9	4.0	6.3	8.4	11.1	17.0	81.0	10.1	7.4
90130	Edmonton	588	0.4	4.2	6.4	8.7	11.6	21.0	56.3	10.8	7.6
90204	Calgary	145	2.0	6.0	9.0	11.0	15.0	28.0	52.0	14.3	9.8
90227	Calgary	472	1.8	4.0	6.0	8.1	11.0	17.0	42.0	9.8	6.4
100106	Vancouver	109	3.0	7.0	9.0	13.0	18.0	39.0	72.0	17.3	12.6
100111	Vancouver	597	2.1	6.0	9.0	12.0	16.0	24.6	62.0	14.1	8.8
100118	Vancouver	343	1.3	5.2	8.0	11.0	14.0	24.0	49.0	12.9	8.0
100303	Victoria	508	0.5	3.6	5.7	8.1	12.0	22.0	59.8	10.7	8.0

Figure 5.13 provides the interquartile range of $PM_{2.5}$ by year, month and day of week for selected sites (urban sites with most complete data record plus rural sites). The seasonal variability of $PM_{2.5}$ is even more pronounced than that of PM_{10}. The Montréal, Ottawa, Edmonton, Calgary and Vancouver/Victoria sites record higher $PM_{2.5}$ concentrations in the winter months and in particular during January and February. The Ontario sites record the highest concentrations in the summer months with a peak median in August. The Maritime sites show variable seasonal variation in $PM_{2.5}$ concentrations with Saint John and Kejimkujik showing a strong summer maximum and Halifax a winter maximum. Most urban sites show minimum $PM_{2.5}$ concentrations on Sunday with maximum concentrations usually occurring during the middle of the week. Using results for all sites, mean mid-week (Tuesday/Wednesday) $PM_{2.5}$ concentrations were 23% higher than Sunday means. For the roadway sites, the differences were larger – up to a 60% increase in $PM_{2.5}$. This indicates that there are large day-of-the-week differences in anthropogenic emissions and significant contributions from motor vehicles.

Yearly variations in $PM_{2.5}$ concentrations during the 1984 to 1995 sampling period (1995 incomplete for most sites) are also shown in Figure 5.13. There is an apparent decrease in $PM_{2.5}$ concentrations at most sites with a complete data record, with the largest percentage decreases occurring at the Montréal-Duncan/Décarie, Edmonton and Vancouver-Rocky Point sites. A trend analysis of $PM_{2.5}$ data carried out by Dann, 1994 for the period 1984-93 showed a statistically significant ($p < 0.001$) decreasing trend in $PM_{2.5}$ on a national basis averaging 3.3% per year. For the Ontario sites, there was no significant change in $PM_{2.5}$ between 1987 and 1993.

$PM_{2.5}$ concentrations are more spatially homogeneous than PM_{10}. There is still significant site to site differences even within the same urban area. The effect of being located near a busy traffic area is exhibited by the roadway site in Montréal (Duncan/Décarie). $PM_{2.5}$ concentrations at this site are higher and more variable than at the downtown site (Ontario Street). This suggests that the vehicles near Duncan St. are contributing a substantial amount of primary and/or secondary fine particles leading to an increase in mean annual $PM_{2.5}$. The magnitude of this vehicular/roadway source is largest in the winter with an increase in mean $PM_{2.5}$ of 4.6 $\mu g/m^3$. In summer the difference is only 1.5 $\mu g/m^3$ (matched sampling days). Such within-city differences are not unique to Montréal. For corresponding sampling days during 1995, a roadway site in Toronto (Evans Ave.) recorded mean $PM_{2.5}$ concentrations that were 3.4 $\mu g/m^3$ higher than the downtown Toronto site (Bay and Grosvenor).

Figure 5.13 The Interquartile Range of $PM_{2.5}$ Concentrations (µg/m³) at National Network Sites by Year, Month and Day of Week. The Plots Indicate the Median, 25th and 75th Percentiles.

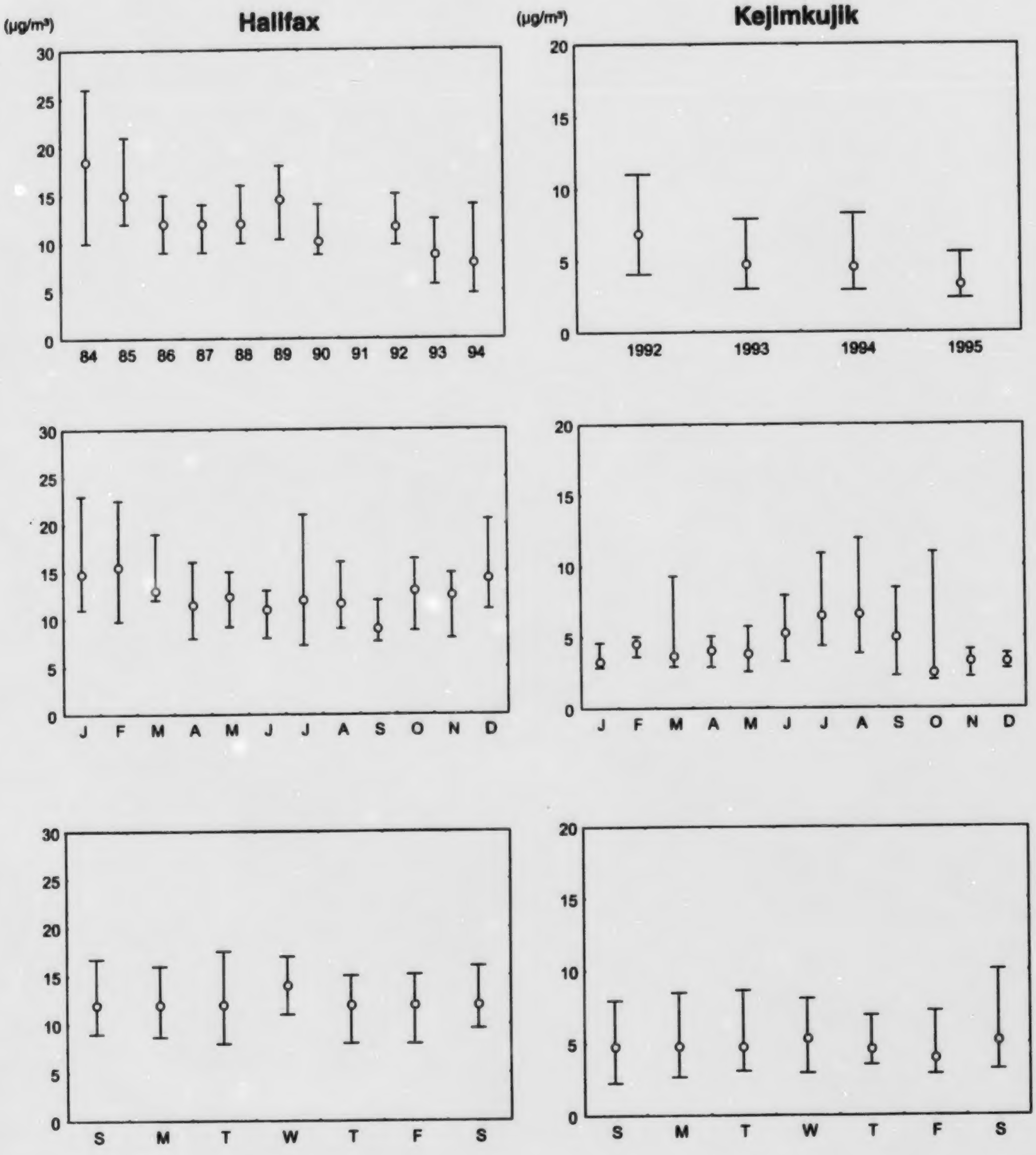


Figure 5.13 The Interquartile Range of $PM_{2.5}$ Concentrations (µg/m³) at National Network Sites by Year, Month and Day of Week. The Plots Indicate the Median, 25th and 75th Percentiles. *(continued)*

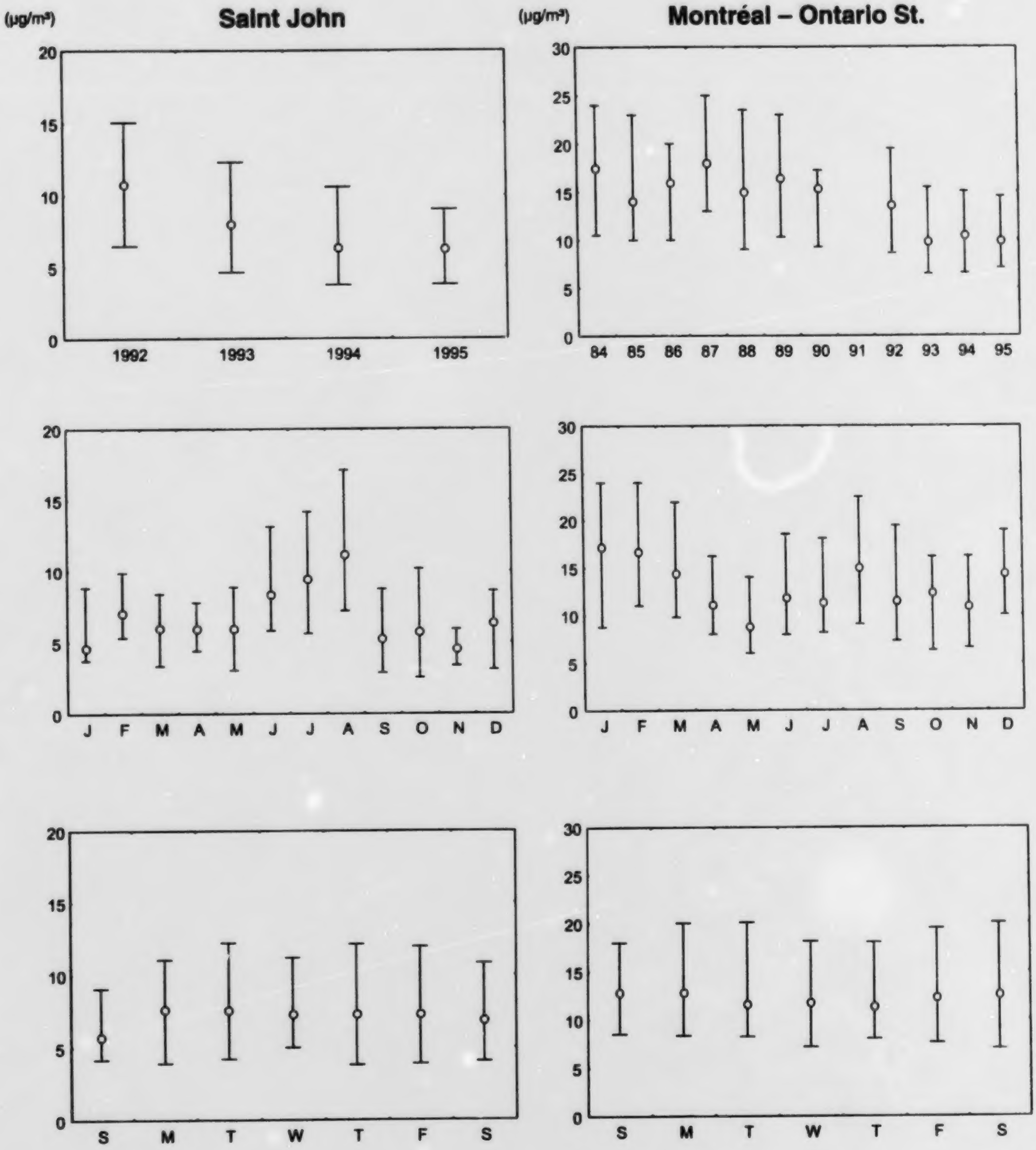


Figure 5.13 The Interquartile Range of $PM_{2.5}$ Concentrations (µg/m³) at National Network Sites by Year, Month and Day of Week. The Plots Indicate the Median, 25th and 75th Percentiles. *(continued)*

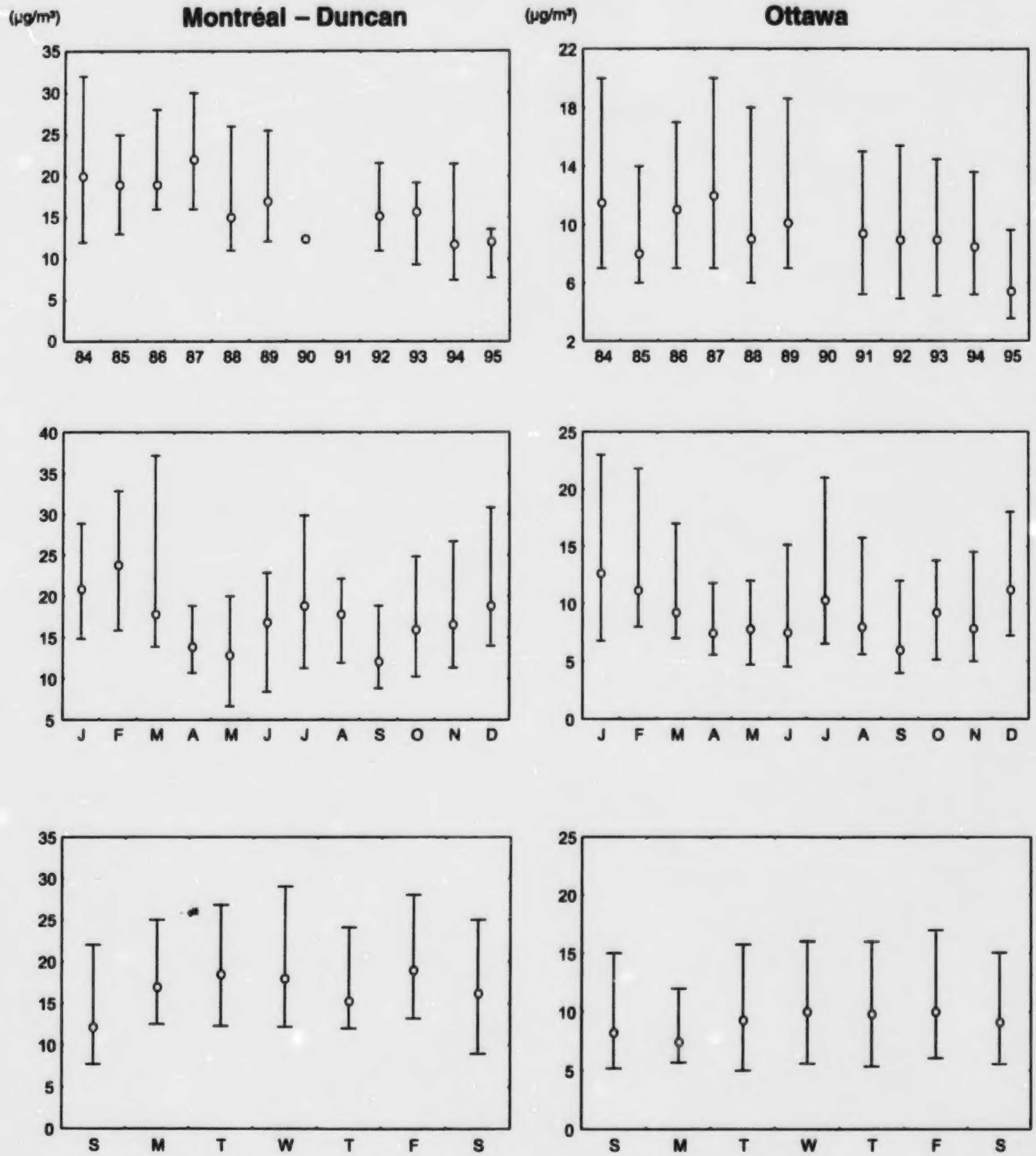


Figure 5.13 The Interquartile Range of $PM_{2.5}$ Concentrations (µg/m³) at National Network Sites by Year, Month and Day of Week. The Plots Indicate the Median, 25th and 75th Percentiles. *(continued)*

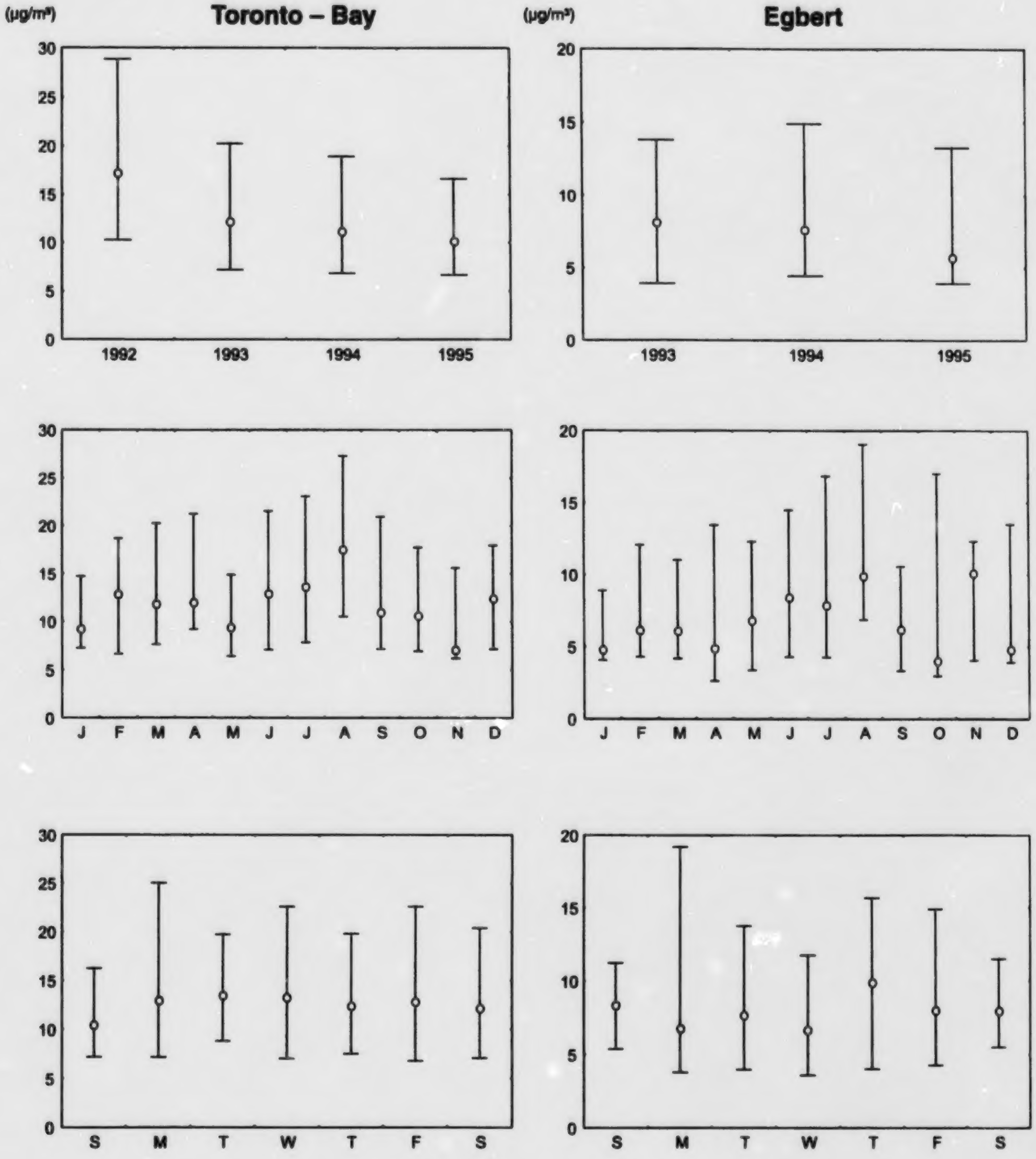
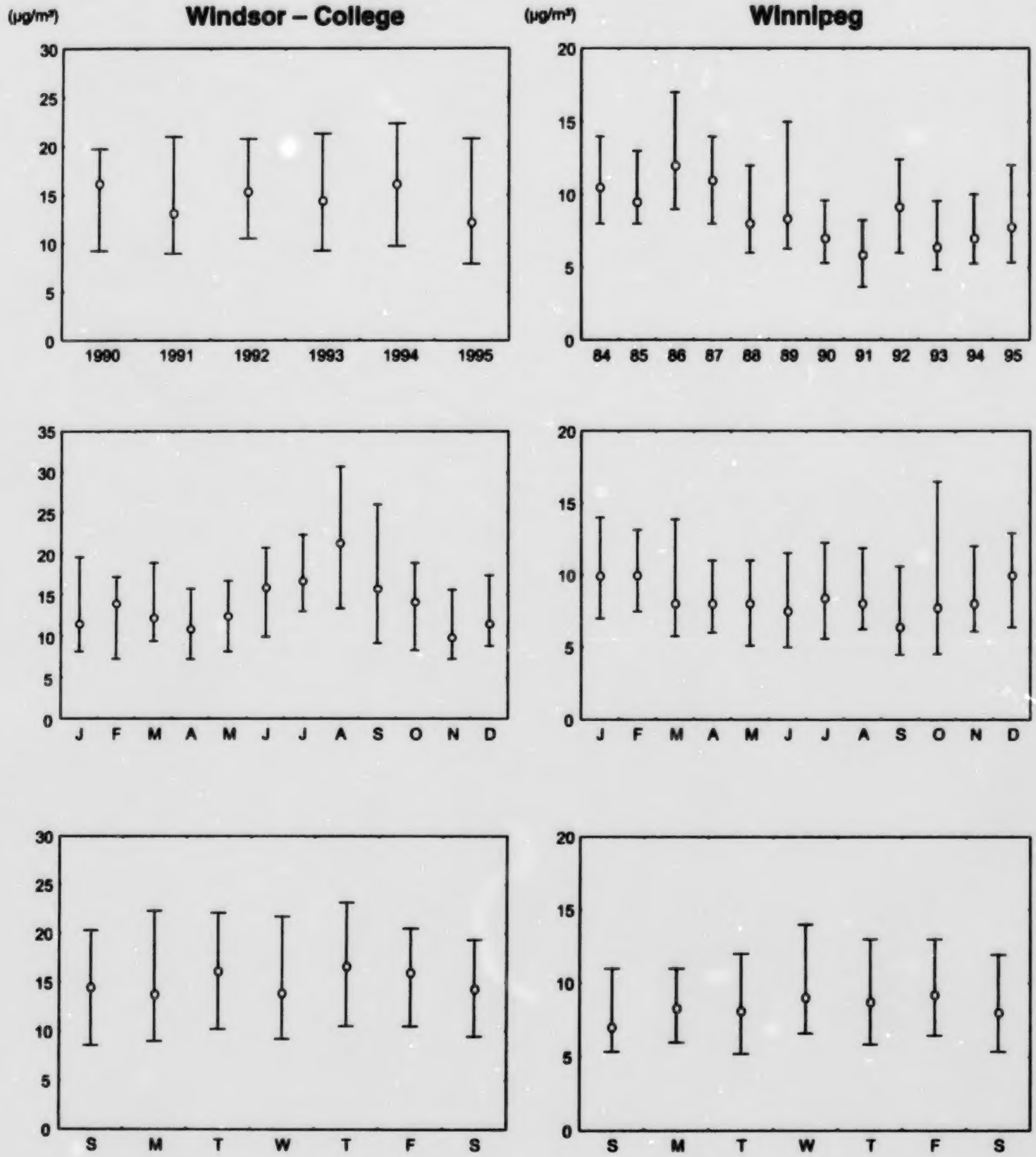


Figure 5.13 The Interquartile Range of PM$_{2.5}$ Concentrations (µg/m³) at National Network Sites by Year, Month and Day of Week. The Plots Indicate the Median, 25th and 75th Percentiles. *(continued)*



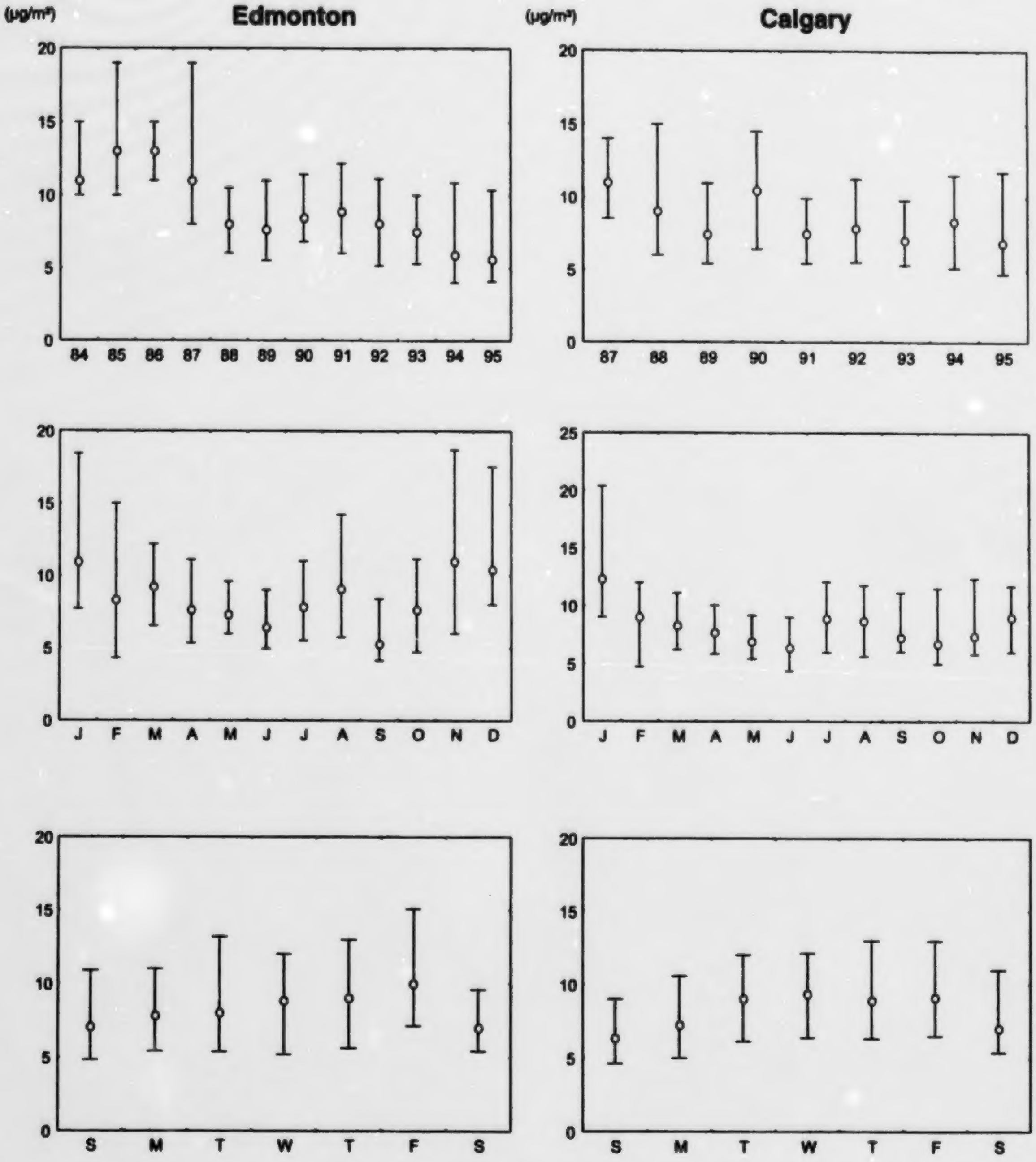


Figure 5.13 The Interquartile Range of $PM_{2.5}$ Concentrations (µg/m³) at National Network Sites by Year, Month and Day of Week. The Plots Indicate the Median, 25th and 75th Percentiles. *(continued)*

Figure 5.13 The Interquartile Range of PM$_{2.5}$ Concentrations (µg/m³) at National Network Sites by Year, Month and Day of Week. The Plots Indicate the Median, 25th and 75th Percentiles. *(continued)*

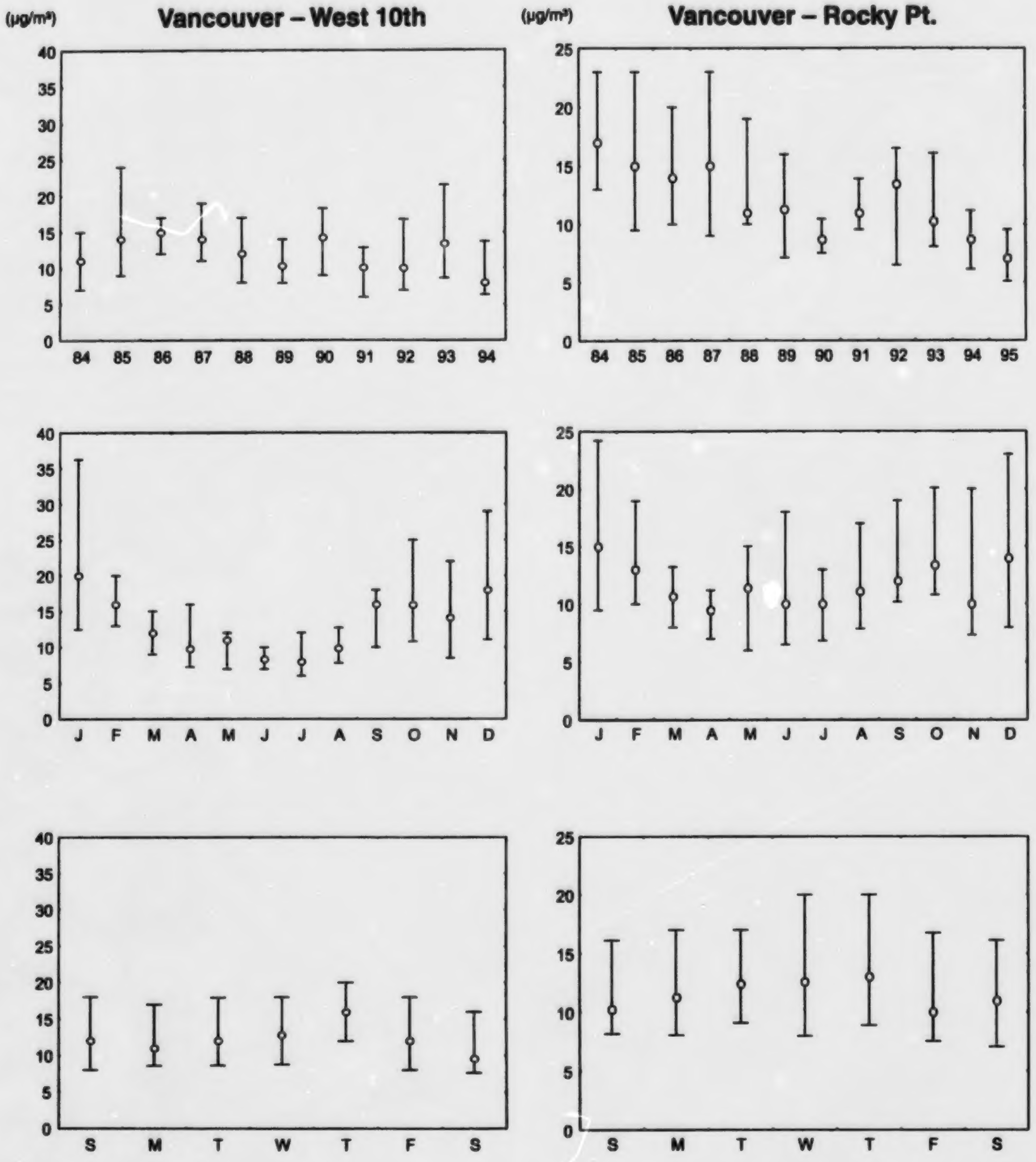
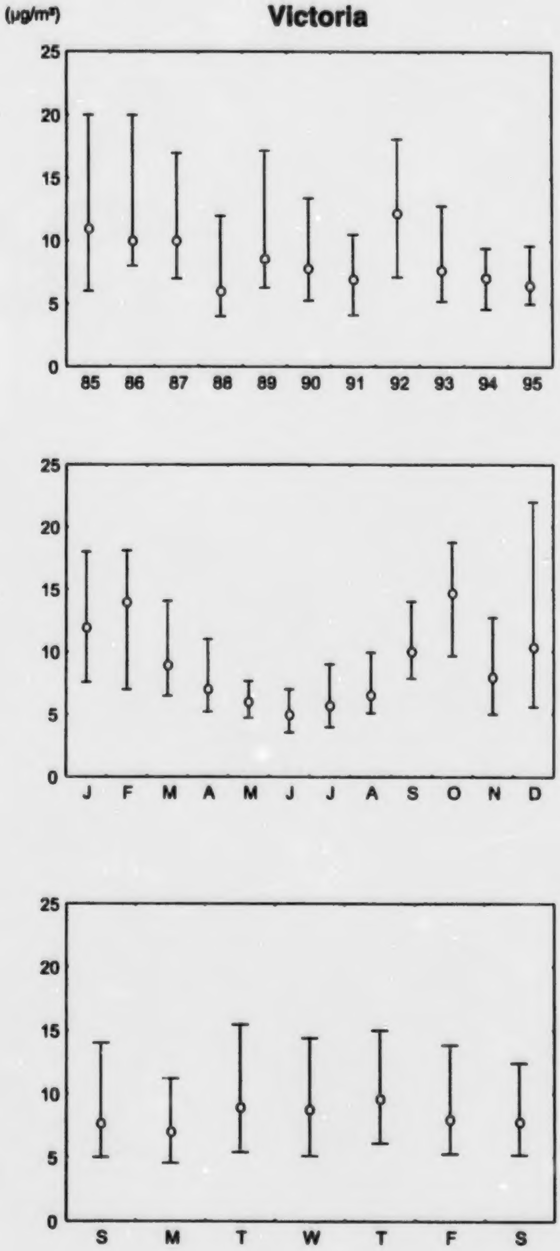


Figure 5.13 The Interquartile Range of $PM_{2.5}$ Concentrations (µg/m³) at National Network Sites by Year, Month and Day of Week. The Plots Indicate the Median, 25th and 75th Percentiles. ***(continued)***



British Columbia

Available $PM_{2.5}$ data from the province are summarized in Table 5.17. $PM_{2.5}$ is currently monitored at sites in Prince George, Quesnel, Williams Lake and Chilliwack using various sampling techniques. Short-term monitoring studies have previously been conducted in Vernon and Nakusp. Additional $PM_{2.5}$ data are available from ten locations in southwestern BC which were part of the REVEAL study conducted during the summer of 1993 (Sakiyama, 1994).

Due to the limited amount of data available, inter-site comparisons are difficult to make. Mean concentrations ranged from 7-13 µg/m³ at those sites having at least one year of data (i.e., Chilliwack and Prince George). At the REVEAL sites, where sampling was limited to the summer months, mean concentrations ranged from 4-11 µg/m³. Observed concentrations were substantially higher in Vernon and Nakusp (18-23 µg/m³), where measurements were made during the winter/spring and fall months, respectively. The maximum observed 24 h concentration of 54 µg/m³ was reported for Prince George.

The frequency of $PM_{2.5}$ data within selected concentration ranges is provided in Table 5.18. Those sites located in the eastern end of the Lower Fraser Valley reported few values greater than 20 µg/m³ (24 h average). In contrast, $PM_{2.5}$ concentrations exceeded 20 µg/m³ over 10% of the time at the Prince George site. At the Vernon and Nakusp sites, which operated less than one year, the frequency of $PM_{2.5}$ concentrations greater than 20 µg/m³ rose to greater than 30 and 50%, respectively.

Table 5.17 Summary of 24 h Average $PM_{2.5}$ Concentrations (µg/m³) – British Columbia

Site No.	Location	Monitor Type[a]	Start Date	Last Date[b]	No. of Samples	% Data Available	Min.	Max.	Mean	Std. Dev.
0450307	Prince George Plaza	Partisol	5-Sep-94	26-Jul-96	115	100	2.0	54.0	13.1	8.9
0500827	Vernon Air	Partisol	23-Dec-95	17-May-96	72	100	2.0	49.0	18.1	11.5
E208096	Quesnel Sr. Sec.	TEOM/Accu	29-Nov-95	26-Jul-96	40	100	2.0	39.0	11.4	5.9
E221197	Williams Lake	Partisol	4-Oct-94	26-Jul-96	97	88	2.0	44.0	10.4	7.1
E222142	Nakusp High School	Partisol	24-Sep-95	23-Dec-95	11	73	7.0	44.0	23.1	12.4
E220891	Chilliwack Airport	TEOM	1-Jun-95	26-Jun-96	383	98	2.3	26.9	7.4	3.3
REVEAL	Island West	IMPROVE	18-Jul-93	15-Aug-93	29	100	1.6	11.0	5.7	2.5
REVEAL	Island East	IMPROVE	18-Jul-93	15-Aug-93	21	71	2.7	14.6	5.9	2.6
REVEAL	UBC	IMPROVE	18-Jul-93	15-Aug-93	29	100	4.4	18.0	9.1	3.5
REVEAL	White Rock	IMPROVE	18-Jul-93	15-Aug-93	28	97	4.7	17.0	8.7	3.3
REVEAL	Langley	IMPROVE	18-Jul-93	14-Aug-93	27	96	3.5	15.5	8.8	3.3
REVEAL	Pitt Meadows	IMPROVE	18-Jul-93	12-Aug-93	22	84	2.6	18.0	8.1	3.7
REVEAL	Clearbrook	IMPROVE	18-Jul-93	15-Aug-93	29	100	2.4	21.8	11.4	4.7
REVEAL	Chilliwack	IMPROVE	19-Jul-93	15-Aug-93	27	96	3.1	20.7	9.8	4.2
REVEAL	Agassiz	IMPROVE	18-Jul-93	15-Aug-93	29	100	3.2	22.6	10.1	5.2
REVEAL	Coquihalla	IMPROVE	18-Jul-93	15-Aug-93	28	97	1.2	8.6	4.3	2.1

[a] *Partisol* refers to a low-volume manual sampler. *TEOM* refers to a continuous monitor. The *TEOM/ACCU* system refers to a filter-based sampling system operating off the TEOM bypass line.

[b] *Last Date* refers to last date for which validated data is available, effective 97/01/31.

Table 5.18 Distribution of $PM_{2.5}$ Concentrations – British Columbia

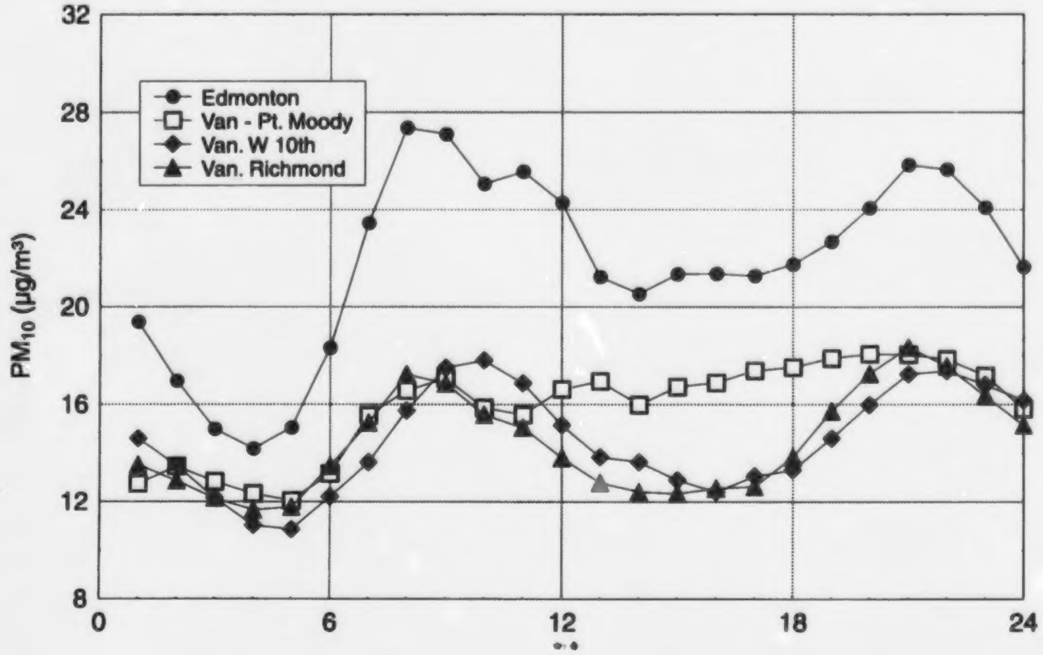
Site I.D.	Name / Location	No. of Samples With Conc. (in μg/m³)					
		>=0	>10	>20	>30	>40	>50
REVEAL	White Rock	28	9	0	0	0	0
REVEAL	UBC	29	9	0	0	0	0
REVEAL	Pitt Meadows	22	5	0	0	0	0
REVEAL	Langley	27	8	0	0	0	0
REVEAL	Island West	29	2	0	0	0	0
REVEAL	Island East	21	2	0	0	0	0
REVEAL	Coquihalla	28	0	0	0	0	0
REVEAL	Clearbrook	29	15	3	0	0	0
REVEAL	Chilliwack	27	12	1	0	0	0
REVEAL	Agassiz	29	11	3	0	0	0
0450307	Prince George Plaza 400	115	66	14	5	3	2
0500827	Vernon Air – RCMP Bldg	72	51	25	13	5	0
E208096	Quesnel Sr. Sec. School	40	24	2	1	0	0
E221197	NW Energy – Williams Lake	97	37	5	3	1	0
E222142	Nakusp High School	11	10	6	3	2	0
E220891	Chilliwack Airport	383	65	3	0	0	0

5.3.5 One-Hour Averaged Data PM_{10} Data

National Network (NAPS)

In 1994, ten sites (all but two in the Lower Fraser Valley) reported hourly PM_{10} concentrations using TEOM instruments. Summary statistics for these sites are shown in Table 5.19. The ratio of 1 h maxima to 24 h maxima ranged from 1.5 to 5. A maximum 1 h PM_{10} concentration of 255 µg/m³ was measured at the Abbotsford site and a maximum 1 h concentration of 204 µg/m³ at the Edmonton site. Diurnal variations in PM_{10} are provided for four of these sites in Figure 5.14. The Figure shows that a substantial increase in PM_{10} levels occurs during the morning rush hour with a minimum at most sites during the mid-afternoon and a secondary peak during the late evening. This is very similar to the diurnal profile measured in Cranbrook, BC as discussed below.

Figure 5.14 Diurnal Variation in PM_{10} Concentrations from Selected NAPS TEOM Monitoring Sites (1994).



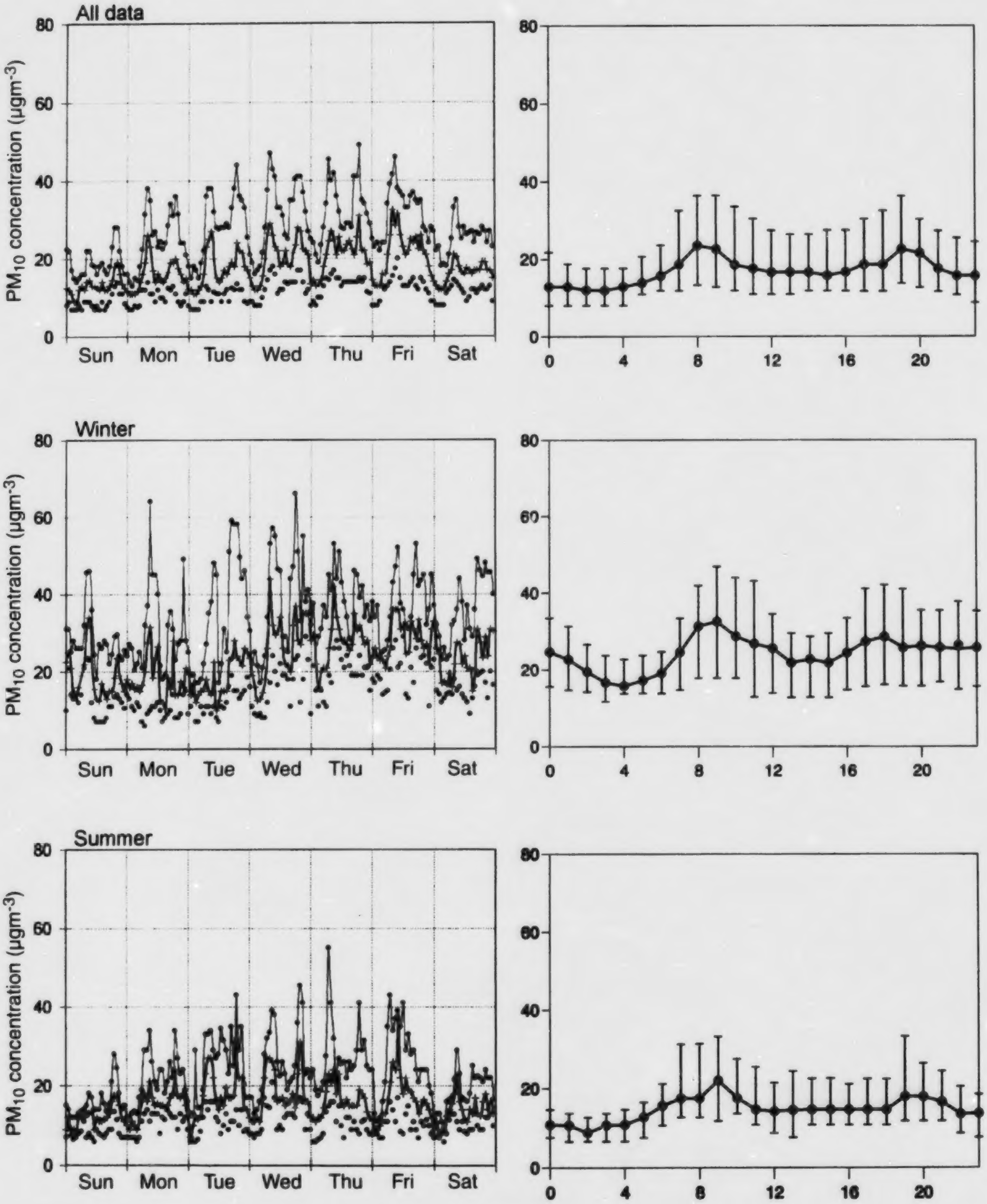
British Columbia

Only one site from the BC database made available hourly averaged PM_{10} data from a period of more than 24 months. This data record is from a site at Cranbrook (E206243), which commenced data acquisition in March 1992. Because of the short duration of this record, it was examined only with regard to diurnal and hebdomadal cycles and the seasonality of these cycles (Figure 5.15). It is clear from Figure 5.15 that in both the summer (July, August, September) and winter (December, January, February) months, the diurnal variability of PM_{10} concentrations is characterized by two periods of elevated concentrations: one during the early morning (8-10 a.m.) and one later in the day (from about 5 p.m. into the night). The former may be indicative of emissions during the morning rush-hour period. The latter may be related to wood burning in residential heating and late afternoon/early evening rush-hour traffic. In the absence of chemical speciation of the aerosols, these sources are speculative. However, the relative intensity of the nocturnal maximum during the winter supports wood burning and evening traffic. Interestingly, the weekday maximum of PM_{10} (especially during Wednesday to Friday/Saturday) is marked during the winter months but is much less evident in the summer data.

Table 5.19 Summary Statistics for Hourly PM_{10} Measurements (TEOM Instruments) – NAPS Network, 1994.

NAPS ID	City	Location		MIN	Percentiles							MAX	% Data	Annual Mean	Std. Dev.
					10	30	50	70	90	95	99				
070119	Winnipeg	65 Ellen Street	1 HR	0	7	14	20	28	45	55	76	99	56	–	–
			24 HR	3	12	17	21	27	38	43	54	61	56		
090122	Edmonton	127 St. & 133 Avenue	1 HR	0	6	11	17	25	45	55	80	204	88	22	17
			24 HR	4	9	14	20	26	37	44	60	75	88		
100110	Vancouver	6400 E. Hastings & Kensington	1 HR	0	5	9	12	16	25	30	41	126	63	–	–
			24 HR	3	7	10	13	16	23	26	31	40	63		
100111	Vancouver	Moody/ Esplanade, Port Moody	1 HR	0	6	11	15	20	30	35	47	89	96	17	10
			24 HR	4	8	12	16	20	28	31	36	40	96		
100118	Vancouver	2550 West 10th Avenue	1 HR	1	6	10	13	18	26	31	43	68	95	15	9
			24 HR	3	8	11	14	17	23	26	31	41	95		
100125	Vancouver	8544 116th Ave., Delta	1 HR	0	5	9	13	18	27	32	45	98	97	15	9
			24 HR	4	8	10	13	17	24	28	35	44	98		
100127	Vancouver	19000 & 72nd Ave., Surrey	1 HR	0	6	9	12	16	24	28	39	74	96	14	8
			24 HR	4	7	10	13	16	21	25	29	35	95		
100128	Vancouver	Williams & Aragon, Richmond	1 HR	1	6	9	12	17	26	32	45	89	98	14	9
			24 HR	4	8	11	14	17	22	25	31	43	99		
101002	Abbotsford	33660 South Fraser Highway	1 HR	0	6	11	17	24	38	47	77	255	44	–	–
			24 HR	3	9	14	20	25	34	40	49	53	44		
101301	Langley	23752 52nd Avenue	1 HR	0	5	9	12	16	24	29	39	65	97	14	8
			24 HR	3	7	10	13	16	21	25	30	35	97		

Figure 5.15 The Interquartile Range and Median PM_{10} Concentrations (µg/m³) at Cranbrook, BC by Day of Week and by Hour of Day for All Data, Winter (D,J,F) and Summer (J,J,A).



5.4 RELATIONSHIPS AMONG TSP, PM_{10} AND $PM_{2.5}$ AND INORGANIC CONSTITUENTS OF PARTICULATE MATTER IN CANADA

The following discussion is drawn largely from Brook et al., 1997.

5.4.1 TSP, PM_{10} and $PM_{2.5}$ and SO_4^{2-}

Fourteen urban sites in the NAPS dichotomous sampler network operating from 1986 to 1994 had simultaneous measurements of TSP, PM_{10}, $PM_{2.5}$ and SO_4^{2-} and combined summary statistics (only for days with all data available) are provided in Table 5.20. For this data set, daily TSP concentrations ranged from 10 to 575 µg/m³ with a majority of the concentrations between 10 and 100 µg/m³. PM_{10} concentrations ranged from 5 to 175 µg/m³; most of the concentrations were below 50 µg/m³. Most of the 24 h $PM_{2.5}$ measurements were between 2 and 30 µg/m³. Overall, the mass distributions for the fine and coarse particles were very similar suggesting that neither mode in the PM_{10} size range dominated the mass distribution. As expected, a large majority of the SO_4^{2-} was found on the fine particles.

Summary statistics for the daily ratios of PM_{10}/TSP, $PM_{2.5}$/TSP, $PM_{2.5}/PM_{10}$, $PM_{2.5-10}/PM_{10}$ and Fine $SO_4^{2-}/PM_{2.5}$ are also provided in Table 5.20. On average, mean PM_{10} accounted for 50% of the TSP, while mean $PM_{2.5}$ accounted for 25% of TSP. The maximum PM_{10}/TSP and $PM_{2.5}$/TSP values were greater than one, which is not expected given that both $PM_{2.5}$ and PM_{10} are fractions of TSP. Errors of this nature are not surprising given the complications of operating an extensive monitoring program involving different agencies. The fact that the dichotomous sampler is a more precise instrument compared to the high volume sampler, and that the maximum $PM_{2.5}/PM_{10}$ value was 1.0, suggests that the outliers are likely due to invalid TSP measurements. However, these samples were not neglected because it could bias the statistics (i.e., there were likely to have been TSP measurements that were larger than the actual concentration, but they could not be identified). Consistent with the similarities in their distributions, fine and coarse particles accounted for equal portions (~50%) of the PM_{10}. Sulphate was, on average across the urban sites, only responsible for 17% of the $PM_{2.5}$. The relationships between TSP, PM_{10}

Table 5.20: Combined Summary Statistics for 14 Urban NAPS Sites in Operation between 1986 and 1994. Sample size, n=2831, is based upon the number of 24 h Periods with Valid Measurements for TSP, PM_{10}, $PM_{2.5}$ and SO_4^{2-}. (FSO_4 and CSO_4 correspond to fine and coarse SO_4^{2-}, respectively)

Measurement	Percentiles					Statistics				
	10	50	70	90	95	Max.	Mean*	S.D.*	Mean#	S.D.#
TSP	22.0	46.0	62.0	98.0	123.0	572.0	55.2	37.8	46.2	1.8
PM_{10}	11.3	24.0	32.0	47.4	58.0	175.0	27.6	16.3	23.6	1.8
$PM_{2.5}$	5.0	11.0	16.0	26.1	32.2	89.0	13.9	9.5	11.4	1.9
Coarse	4.4	11.0	15.4	25.4	33.0	87.0	13.7	10.4	10.7	2.0
FSO_4	0.6	1.9	3.1	6.0	8.8	39.0	2.9	3.3	1.9	2.4
CSO_4	0.1	0.3	0.4	0.6	0.9	4.6	0.4	0.3	0.3	2.2
Ratios										
PM_{10}/TSP	0.30	0.51	0.64	0.87	1.00	2.40	0.56	0.24	0.51	1.52
$PM_{2.5}$/TSP	0.10	0.26	0.37	0.54	0.64	1.41	0.30	0.19	0.25	1.91
$PM_{2.5}/PM_{10}$	0.29	0.50	0.60	0.74	0.80	1.00	0.51	0.17	0.48	1.45
Coarse/PM_{10}	0.26	0.50	0.58	0.71	0.76	1.00	0.49	0.17	0.46	1.51
$FSO_4/PM_{2.5}$	0.07	0.19	0.25	0.36	0.42	0.95	0.20	0.11	0.17	1.89

* arithmetic average and arithmetic standard deviation (S.D.)

# geometric mean and geometric standard deviation (S.D.)

and $PM_{2.5}$ are dependent on concentration. Ratios of PM_{10} to TSP and $PM_{2.5}$ to TSP decrease with increasing TSP concentrations as a greater proportion of the TSP mass is represented by very coarse particles. As shown in Table 5.21, an exponential equation best fits the data sets.

Table 5.21: Relationships between TSP, PM_{10} and $PM_{2.5}$ (using data from 14 Urban sites)

Equation	r^2
Linear Relationship	
* PM_{10} = 0.44 · TSP	0.817
$PM_{2.5}$ = 0.73 + 0.49 · PM_{10}	0.636
* $PM_{2.5}$ = 0.21 · TSP	0.620
Exponential Relationship	
* PM_{10} = 10** (0.826 · log TSP)	0.986
$PM_{2.5}$ = 10** (-0.25 + 0.949 · log PM_{10})	0.668
* $PM_{2.5}$ = 10** (0.636 · log TSP)	0.946

* Intercepts forced to zero

TSP, PM_{10}, $PM_{2.5}$ and SO_4^{2-} mass distributions for a cross-section of Canadian cities are compared in the box plots in Figure 5.16. Only days with observations of all four parameters were used to allow a proper comparison between parameters at each site. Each of the sites has a relatively long data record ($n > 220$ days; actual n depends upon site). Sites in the three Prairie cities of Winnipeg, Calgary and Edmonton have large and variable TSP concentrations, but their $PM_{2.5}$ concentrations are small relative to the other sites and exhibit less variability. As discussed previously, much of the airborne PM observed in these areas is mechanically derived and likely consists of local crustal material.

There is an obvious decrease in the SO_4^{2-} levels from the sites located east of the upper Great Lakes to those located west of the lakes. This is a direct reflection of the magnitude and spatial density of SO_2 emissions within and upwind of these two areas. SO_4^{2-} concentrations are highest in southern Ontario although the mean levels in µg/m³ are surprisingly similar from Ontario to Halifax, NS.

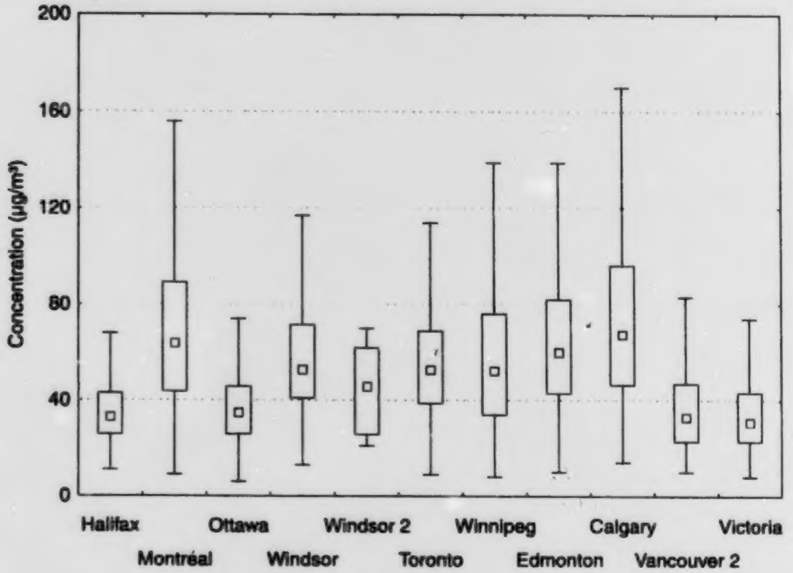
Mean $PM_{2.5\text{-}10}$, $PM_{2.5}$ and SO_4^{2-} concentrations were calculated separately for winter and summer periods and are displayed for selected sites in Figures 5.17. All available data for each site were used (i.e., sample size differs for parameters) and locations representing rural eastern Canada are included. As discussed in the preceding sections, summer mean $PM_{2.5}$ levels are higher over eastern Canada compared to the west. The reason for this difference is that in the summertime SO_4^{2-} increases dramatically at the eastern sites. East to west differences in mean $PM_{2.5}$ levels are less in the winter due to decreases in the eastern concentrations of SO_4^{2-} and to relatively large increases in $PM_{2.5}$ in Vancouver and Victoria. With the exception of Toronto and Windsor, all the sites shown have higher $PM_{2.5}$ levels in the winter, but the differences are greatest at Vancouver and Victoria. A number of factors could be leading to this predominant wintertime increase in $PM_{2.5}$. Mixing heights are usually lower and energy consumption for heating is higher. Wood burning is also more prevalent.

On average, increases in PM_{10} from summer to winter are generally equal to or less than the magnitude of the $PM_{2.5}$ increases. Therefore, the additional PM_{10} found in the winter is likely due to fine particles and not coarse particles. This suggests that combustion sources are an important contributor to wintertime particle pollution and shows that fine particles drive the seasonal PM_{10} variability. The Toronto, Windsor and Winnipeg sites are the only ones included in Figure 5.17 where PM_{10} clearly decreases from summer to winter. At Winnipeg, this is due to a decrease in coarse particles, while at Toronto and Windsor both the coarse and fine particle levels drop slightly.

On average, coarse particles decrease from summer to winter at most of the sites shown. This may be due to snow cover and/or increases in precipitation, which would inhibit the suspension of crustal material. There are visible differences in the mean summer and winter $PM_{2.5}$ levels, but there is a large amount of overlap in the mass distributions with a similar amount of variability in both seasons. The sites with the largest differences in mean $PM_{2.5}$ between the summer and winter are Saint John, Kejimkujik, Windsor (both sites) and Walpole. These differences are probably a result of SO_4^{2-} seasonality since at each of these sites, a relatively large fraction of the $PM_{2.5}$ is comprised of SO_4^{2-}. Qualitatively, all sites west of Winnipeg have higher mean and median PM_{10} concentrations in the winter and all of those from Winnipeg east, excluding Ottawa, Montréal-Duncan/Décarie, Québec and Halifax, show the opposite tendency.

Figure 5.16 Comparison of the Distributions of TSP, PM_{10}, $PM_{2.5}$ and SO_4^{2-} at 11 Urban Sites (1984-1993). The box plots indicate the median, 5th, 95th, 25th, and 75th percentiles.

a) TSP



b) PM_{10}

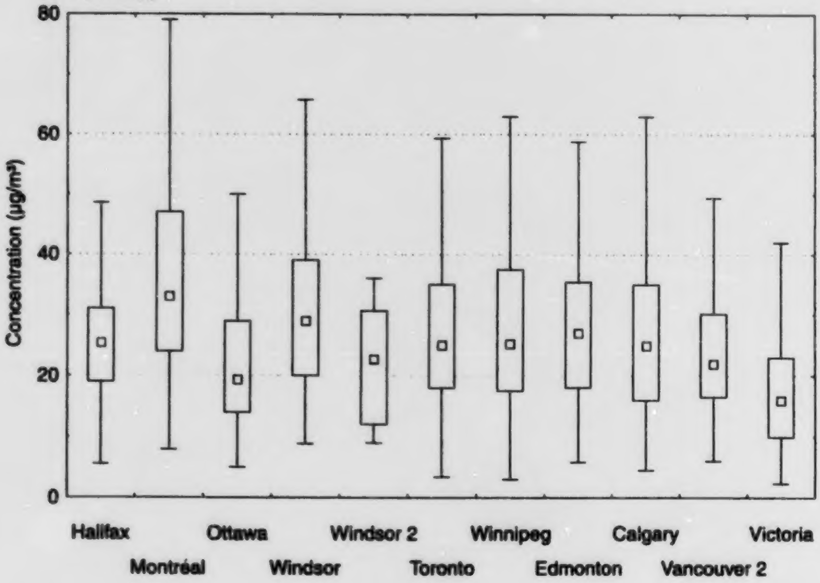
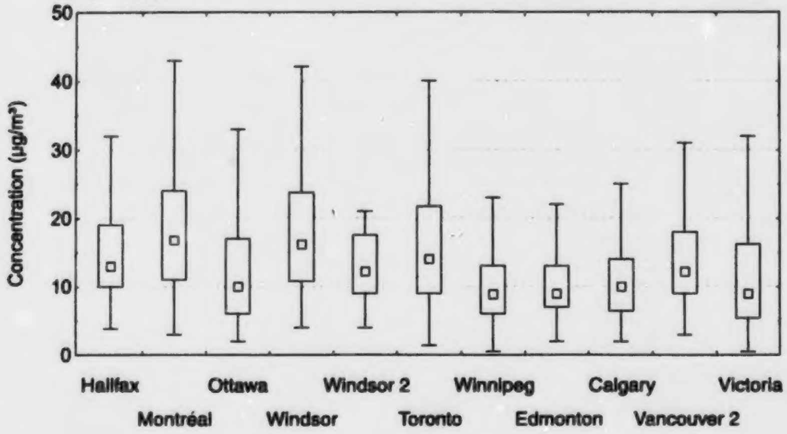
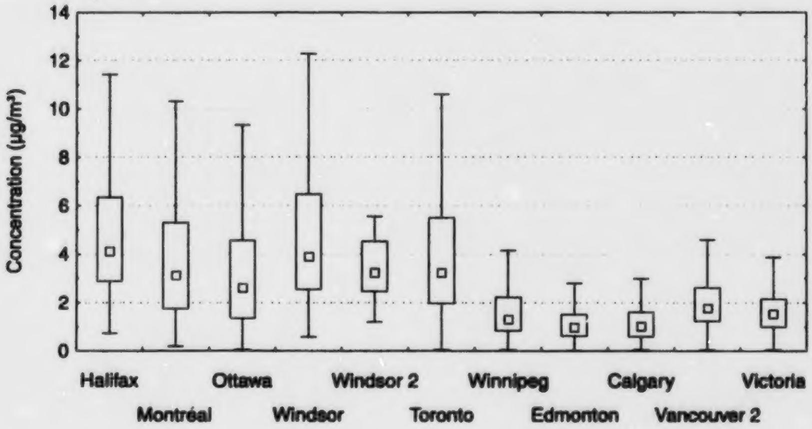


Figure 5.16 Comparison of the Distributions of TSP, PM_{10}, $PM_{2.5}$ and SO_4^{2-} at 11 Urban Sites (1984-1993). The box plots indicate the median, 5th, 95th, 25th, and 75th percentiles. *(continued)*

c) $PM_{2.5}$



d) SO_4^{2-}

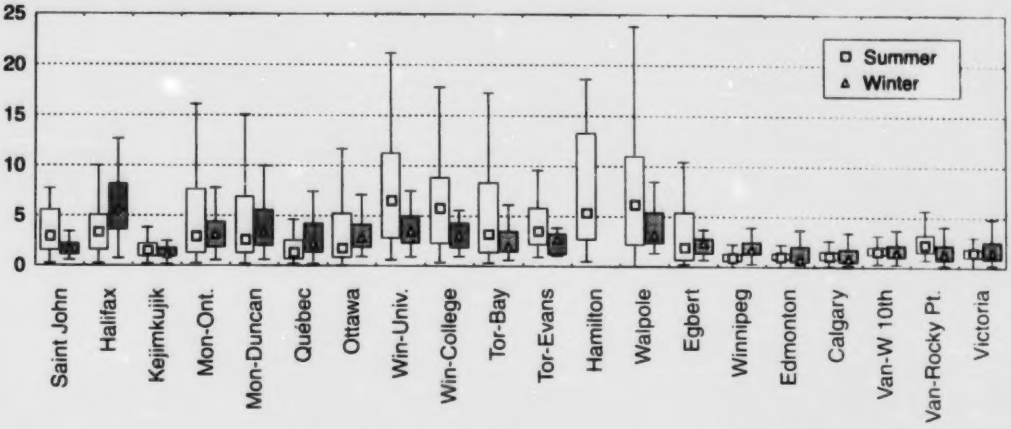


5.4.2 Site-Specific Relationship Between PM_{10} and $PM_{2.5}$

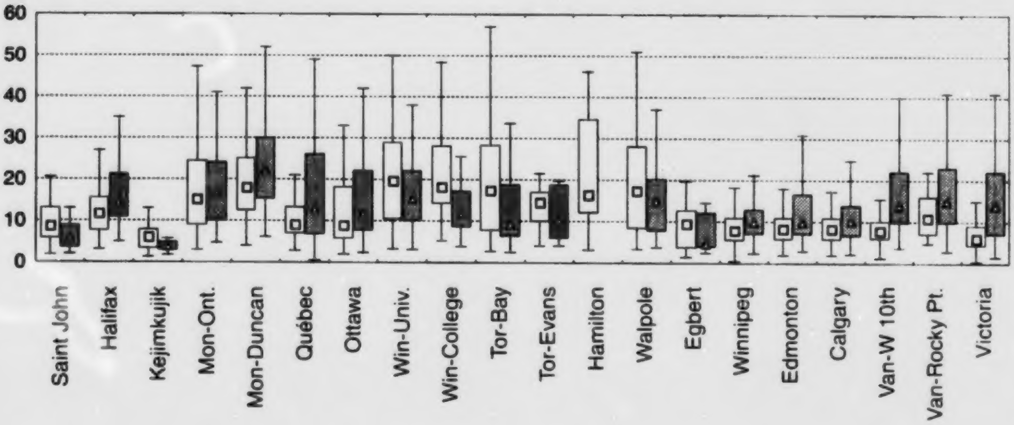
Site by site relationships between fine ($PM_{2.5}$), coarse and PM_{10} mass concentrations are shown for the NAPS Network dichotomous sampler sites in Table 5.22. On average across all sites, 53% of mean PM_{10} is made up of fine particles. However, there is a considerable amount of variability in their ratio among sites and between individual observations. Differences in the $PM_{2.5}$ to PM_{10} ratio across a variety of sites are displayed in Figure 5.18. At the urban locations, the mean ratio ranges from around 0.38 in the Prairies to 0.59 on the West Coast. The mean ratio ranges from 0.60-0.65 at the rural sites. From event to event, there is even more variability. For example, in Toronto the 5th and 95th percentiles of the ratio are about 0.15 and 0.90, respectively. The interquartile range (difference between the 25th and 75th percentiles) for Toronto (combined sites) is close to 0.25. The interquartile range is between 0.15 and 0.25 for most of the sites. This indicates that about 50% of the time the ratio of $PM_{2.5}$ to PM_{10} does not vary by much more than ±0.10 at a given site.

Figure 5.17 Comparison of $PM_{2.5-10}$, $PM_{2.5}$ Mass and SO_4 Concentrations Between Summer (J,J,A) and Winter (D,J,F) at NAPS Dichotomous Sampler Sites. The Box Plots Indicate the Median, 25th and 75th Percentiles and the Non-Outlier Minimum and Maximum Concentrations.

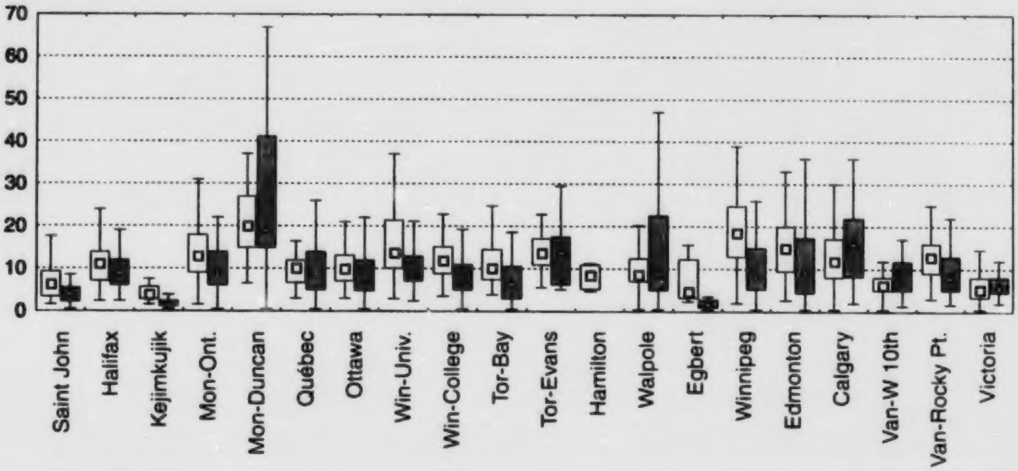
a) SO_4^{2-}



b) Fine



c) Coarse



There are relatively strong correlations (r^2) between PM_{10} and $PM_{2.5}$ at each site (see final column, Table 5.22). R^2 values are greater than or equal to 0.70 at all but the three Prairie sites. As expected, correlations are larger at sites where $PM_{2.5}$ comprises a larger fraction of PM_{10} and/or at sites with stronger fine to coarse correlations. Winnipeg, Calgary and Edmonton have the smallest mean $PM_{2.5}$ to PM_{10} ratios. Accurate predictions of $PM_{2.5}$ from PM_{10} or vice versa are not likely even though day to day changes in PM_{10} or $PM_{2.5}$ are well correlated; there is considerable unexplained variability in their ratios. There are also relatively large standard errors for the regression coefficients.

The strength of the $PM_{2.5}$ to PM_{10} correlations listed in Table 5.22 is consistent with the belief that the temporal variations in $PM_{2.5}$ have a significant influence on the observed variability in PM_{10} (Kao and Friedlander, 1995; Wilson and Burton, 1995). Measures of variability reported in Table 5.22 and the r^2 values associated with the PM_{10} to coarse particle relationship also tend to support this notion. At a majority of the sites, the daily variations in PM_{10} are correlated more with the fine particles than with the coarse particles. Again, the Prairie sites are an exception to this pattern. The coarse particle variations are also of greater potential importance at Montréal-Duncan/Décarie. Coefficient of variations (standard deviations for both the fine and coarse mass divided by the mean PM_{10} concentration) were calculated to assess how much of the PM_{10} variability may be attributable to the variations in fine and coarse particles. These variability measures are larger for the fine than they are for the coarse at a majority of the sites. This does suggest that the fine mass has a greater influence on PM_{10} variability. However, the differences between the fine and coarse fractions are not overwhelming, which indicates that the coarse particles can also contribute substantially to the overall PM_{10} variability. In particular, the coarse mass dominates at the Prairie sites and has the potential to significantly influence the variability in PM_{10} at the Montréal-Duncan/Décarie and Québec City sites. The greater influence by fine particles is most evident at the rural locations and at sites not heavily impacted upon by urbanization (i.e., traffic, industry, and construction).

The statistical results in Table 5.22 indicate that there is some common variability between the fine and coarse size fractions. In general, the correlation (r^2) between the fine and coarse particles is smaller at the sites where the suspension of crustal material is thought to be a more important factor. This includes Winnipeg, Edmonton, and Walpole Island, which is in an area with agricultural activity.

Site by site relationships between $PM_{2.5}$ mass and fine SO_4^{2-} concentrations are shown for the NAPS Network dichotomous sampler sites in Table 5.23. Mean $SO_4^{2-}/PM_{2.5}$ ratios ranged from 0.24 to 0.34 at the eastern sites (excluding Montréal-Duncan/Décarie and Québec City) and from 0.12 to 0.16 at the sites west of Ontario. For sites with complete data, the correlation (r^2) between SO_4^{2-} and $PM_{2.5}$ was highest at the Atlantic and Ontario sites and smallest at the Winnipeg site.

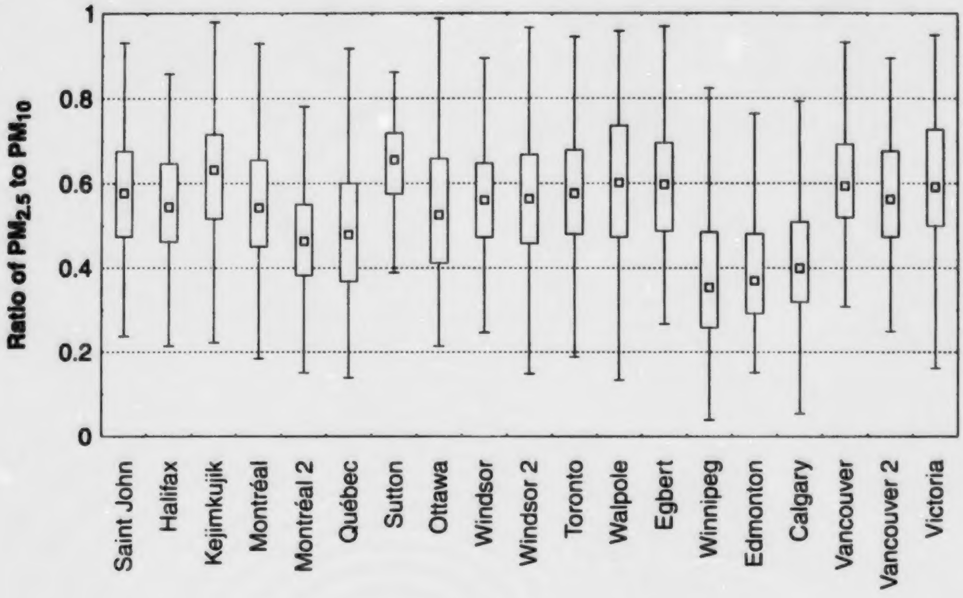
Mean concentrations and correlation coefficients were calculated separately for four pairs of urban-rural sites. Results are shown in Table 5.24. For each pair of sites, only the days when both sites provided valid measurements were used. With the exception of Walpole Island, particle mass concentrations are lower at the rural locations. Walpole Island is 55 km north of Windsor, and is influenced heavily by Detroit, Michigan. Egbert is 50 km north of Toronto, Sutton is 75 km southeast of Montréal and Kejimkujik is 120 km southeast of Saint John. For the other pairs of sites, the urban enhancement in concentration is greatest for the coarse particles. For example, the mean coarse particle concentration is 46% higher in Toronto compared to Egbert, while the mean amount of fine particles is only 28% higher in Toronto. This behaviour stems from the slower rate of deposition of fine particles, which leads to more spatial homogeneity and greater importance of long range transport, which is likely to influence sites in close proximity to one another similarly. Consequently, sites that are close together experience similar fine particle levels, while coarse particle levels differ more between sites. This is also reflected in the urban versus rural correlation coefficients, which are systematically higher for fine particles.

Table 5.22 Site by Site Relationships between Fine, Coarse and PM_{10} Mass (All samples 1984-1993).

Location	Number of Samples	Mean Fine Mass (μg/m³)	Mean Coarse Mass (μg/m³)	Mean $PM_{2.5}$ to Mean PM_{10} Ratio	C.V. for PM_{10}	C.V. for Fine Mass*	C.V. for Coarse Mass*	Coarse to Fine r^2	Coarse to PM_{10} r^2	Linear Equation $PM_{2.5} = a + b \cdot PM_{10}$		
										b	a	r^2
Saint John	292	10.1	7.3	0.58	0.74	0.37	0.29	0.37	0.77	0.58	0.09	0.86
Halifax	304	14.1	11.5	0.55	0.55	0.25	0.23	0.25	0.66	0.55	0.06	0.74
Kejimkujik	277	7.2	4.2	0.63	0.90	0.49	0.34	0.49	0.56	0.69	-0.62	0.86
Montréal	577	15.9	11.9	0.57	0.58	0.30	0.28	0.30	0.55	0.65	-2.12	0.81
Montréal-Duncan/Décarie	314	20.9	23.7	0.47	0.63	0.25	0.33	0.25	0.76	0.46	0.37	0.70
Québec City	221	11.9	11.6	0.50	0.65	0.28	0.35	0.28	0.63	0.54	-0.72	0.70
Sutton	136	7.7	3.5	0.68	0.40	0.40	0.22	0.40	0.66	0.83	-1.59	0.98
Ottawa	358	12.6	10.0	0.56	0.63	0.31	0.31	0.31	0.54	0.63	-1.66	0.77
Windsor	352	18.1	13.8	0.57	0.65	0.32	0.27	0.32	0.71	0.57	-0.16	0.81
Windsor-College	422	16.8	12.9	0.56	0.62	0.28	0.26	0.28	0.56	0.59	-0.76	0.73
Toronto	586	16.8	11.3	0.60	0.64	0.33	0.27	0.33	0.62	0.64	-1.14	0.84
Walpole	275	17.6	12.2	0.59	1.07	0.45	0.35	0.45	0.59	0.56	1.00	0.70
Egbert	137	10.4	6.6	0.61	0.80	0.45	0.31	0.45	0.71	0.64	-0.49	0.89
Winnipeg	447	10.3	18.4	0.36	0.75	0.22	0.48	0.22	0.82	0.29	2.02	0.42
Edmonton	380	10.5	16.4	0.39	0.61	0.19	0.41	0.19	0.76	0.34	1.32	0.46
Calgary	504	11.2	15.6	0.42	0.66	0.24	0.41	0.24	0.79	0.41	0.30	0.65
Vancouver	334	15.5	9.2	0.63	0.52	0.33	0.23	0.33	0.49	0.74	-2.71	0.88
Vancouver-W 10th	360	15.6	11.3	0.58	0.57	0.29	0.25	0.29	0.56	0.65	-1.78	0.81
Victoria	393	11.5	6.3	0.65	0.51	0.37	0.27	0.37	0.36	0.81	-2.91	0.91
All Sites	6669	13.8	12.1	0.53	0.63	0.38	0.37	0.13	0.66	0.52	0.32	0.70

* Coefficient of variation is determined relative to the PM_{10} mean concentration (i.e., Standard Deviation for particle size of interest/mean PM_{10}).

Figure 5.18 Distributions of the Ratio of $PM_{2.5}$ to PM_{10} Mass at the NAPS Dichotomous Sampler Sites. The Box Plots Indicate the Median, 5th and 95th and 25th and 75th Percentiles.



Although sample sizes decrease substantially for the SO_4^{2-} data there appears to be a clear increase in correlation compared to the fine mass correlations. This is due to the strong influence of long-range transport on SO_4^{2-} levels in eastern Canada (i.e., the further the pollutants travel the more they become homogeneously mixed over large areas). Some of the increased correspondence between sites in the fine mass compared to coarse mass is driven by similarity in SO_4^{2-} levels. Conversely, the apparent decrease in correlation from SO_4^{2-} to $PM_{2.5}$ suggests that some of the other fine mass constituents have more localized sources which do not impact evenly on their neighbouring urban or rural sites.

The higher particle concentrations at the urban sites are a reflection of the greater amounts of human activity (e.g., traffic and construction). The paved surfaces and smaller amounts of vegetation probably contribute to the elevated concentrations of coarse particles as well. More unpaved roads, agricultural activity and open areas with large undisturbed fetches should enhance the suspension of crustal material (coarse particles) at the rural sites; these factors do not outweigh contributing to more coarse particles in urban areas.

Table 5.23 Site by Site Relationships between Fine SO_4^{2-} and Fine Mass ($PM_{2.5}$) – All samples 1984-1995.

NAPS ID	City	Address	N	Fine Mass ($PM_{2.5}$) µg/m³	Fine SO_4^{2-} µg/m³	Ratio of Mean SO_4^{2-} to Mean $PM_{2.5}$	Linear Equation $SO_4^{2-} = a + b \cdot PM_{2.5}$		
							b	a	r²
40203	Saint John	Forest Hills	172	8.4	2.8	0.33	0.37	-0.35	0.78
30101	Halifax	N. S. Technical College	310	13.8	4.7	0.34	0.40	-0.72	0.77
30118	Halifax	1657 Barrington St.	90	8.5	3.0	0.35	0.34	0.11	0.66
30119	Dartmouth	Harbor View School	83	6.9	2.3	0.34	0.36	-0.17	0.75
30501	Kejimkujik	National Park	107	6.5	2.2	0.34	0.42	-0.50	0.87
50104	Montréal	1125 Ontario Est	410	17.0	4.1	0.24	0.30	-0.97	0.62
50109	Montréal	Duncan/Décarie	316	20.9	4.0	0.19	0.21	-0.30	0.59
50307	Québec City	Parc Cartier Breboeuf	265	11.5	2.2	0.20	0.18	0.23	0.48
54101	Sutton		24	7.1	2.4	0.34	0.46	-0.87	0.92
60104	Ottawa	Rideau/Wurtemburg	392	12.5	3.4	0.28	0.32	-0.57	0.60
60204	Windsor	471 University Ave.	327	17.6	5.2	0.29	0.38	-1.43	0.76
60211	Windsor	College/Prince	184	15.5	4.5	0.29	0.42	-2.08	0.71
60417	Toronto	26 Breadalbane Street	272	16.7	4.6	0.28	0.36	-1.42	0.72
60424	Toronto	Bay & Grosvenor	187	14.6	3.7	0.25	0.31	-0.81	0.78
60403	Toronto	Evans & Arnold	72	12.6	3.1	0.25	0.33	-1.09	0.61
60512	Hamilton	Egin & Kelly	24	18.0	6.1	0.34	0.44	-1.76	0.92
61901	Walpole Island		236	17.0	5.5	0.32	0.29	0.45	0.64
64401	Egbert		55	8.8	2.7	0.31	0.33	-0.20	0.79
70119	Winnipeg	65 Ellen Street	455	10.3	1.6	0.16	0.06	1.03	0.15
90130	Edmonton	10255-104th Street	405	10.3	1.4	0.13	0.11	0.21	0.39
90204	Calgary	316-7th Ave.	143	14.5	1.8	0.12	0.13	-0.14	0.29
90227	Calgary	611-4th Street	380	10.0	1.3	0.13	0.10	0.34	0.31
100106	Vancouver	2294 West 10th Ave.	106	17.2	2.3	0.14	0.08	0.94	0.50
100111	Vancouver	Rocky Pt. Park	366	15.5	2.0	0.13	0.07	0.89	0.28
100118	Vancouver	2550 West 10th Ave.	295	12.9	1.8	0.14	0.08	0.76	0.41
100303	Victoria	1250 Quadra St.	391	11.6	1.7	0.15	0.10	0.59	0.53

Table 5.24 Direct Comparisons of Urban and Rural Site Measurements of Fine, Coarse and PM_{10} Mass and Sulphate (r corresponds to the Pearson correlation coefficient).

			Coarse Fraction			Fine Fraction			Total (PM_{10})		
Site 1	**Site 2**	**N**	**Site 1 Mean Mass (µg/m³)**	**Site 2 Mean Mass (µg/m³)**	**r**	**Site 1 Mean Mass (µg/m³)**	**Site 2 Mean Mass (µg/m³)**	**r**	**Site 1 Mean Mass (µg/m³)**	**Site 2 Mean Mass (µg/m³)**	**r**
Mass											
Saint John	Kejimkujik	370	7.0	4.0	0.22	10.1	7.8	0.67	17.1	11.8	0.54
Montréal	Sutton	131	8.9	3.5	0.53	11.6	7.6	0.82	20.6	11.2	0.78
Toronto	Egbert	253	9.9	6.2	0.50	14.3	10.8	0.86	24.2	17.0	0.78
Windsor	Windsor 2	193	12.7	11.5	0.49	16.7	15.7	0.81	29.5	27.2	0.70
Windsor	Walpole	249	13.7	13.0	0.26	18.3	18.3	0.58	31.9	31.3	0.50
Windsor 2	Walpole	186	12.2	14.8	0.26	16.6	21.1	0.56	28.7	35.8	0.46
SO_4^{2-}											
Saint John	Kejimkujik	44	0.36	0.17	0.36	3.1	2.3	0.84	3.5	2.5	0.83
Montréal	Sutton	15	0.22	0.15	0.64	3.1	2.8	0.92	3.4	2.9	0.91
Toronto	Egbert	9	0.26	0.30	0.53	5.1	4.7	0.90	5.4	5.0	0.90
Windsor	Windsor 2	111	0.49	0.94	0.49	4.8	4.1	0.90	5.3	5.1	0.92
Windsor	Walpole	161	0.52	0.40	0.51	5.8	5.8	0.71	6.3	6.1	0.73
Windsor 2	Walpole	74	0.89	0.39	0.34	4.2	7.2	0.69	5.2	7.4	0.78

5.4.3 Inorganic Composition of PM_{10} and $PM_{2.5}$

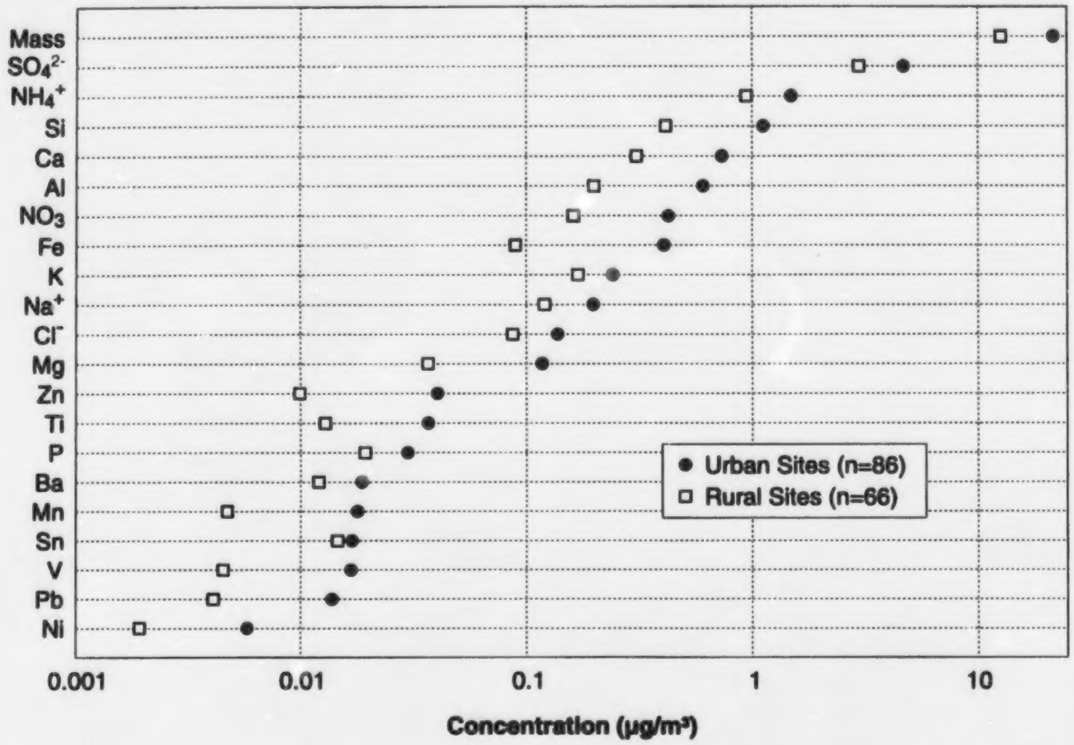
Forty elements and 25 anion and cation species are determined from the fine and coarse particles. Figure 5.19 is a comparison of the mean concentrations of the most abundant inorganic elements and ions measured during May-September 1993 at urban and rural sites (Saint John, Kejimkujik, Montréal, Sutton, Toronto and Egbert). For all components listed in Figure 5.19, the concentrations are higher at the urban sites. There are several elements/ions for which the urban-rural difference is disproportionately greater than the total mass difference, indicating that these constituents are enriched in urban areas. The relative amounts of Ca, Si, NO_3^-, Fe, Al, Mg, Zn, Ti, Mn, V, Pb and Ni are larger in the urban particle samples compared to the rural samples. Some of these species are common in crustal material (e.g., Si, Ca, Ti), indicating greater suspension of dust in urban areas. Industrial and combustion activities are likely responsible for the urban enrichment of some of the other species, such as NO_3^-, V and Pb. In general, it appears that Ba, P and K levels were affected by processes similar to those affecting SO_4^{2-} and NH_4^+ (i.e., long-range transport and slow deposition velocities). In addition, the slightly higher proportion of K could be due to more vegetative sources or to more wood/biomass burning in the rural areas, while the higher proportion of Sn is difficult to explain. The similarity in the relative levels of Na^+ and Cl^- between the urban and rural sites is likely driven by Saint John and Kejimkujik, both of which are situated near the ocean.

Median concentrations for some of the more abundant chemicals are listed in Table 5.25. Only selected cities, representing a range of geographical areas are included. Sulphate and sulphur are the most abundant PM_{10} constituents at all of the sites shown. Al, Ca and/or Si are the next most abundant elements. Some of the chemical differences among sites are consistent with their geographical locations and the sources in the vicinity and upwind of the sites. Median concentrations of Si, Ca, Mg and Fe, which are typically associated with crustal material, tend to be high at the two Prairie sites. Concentrations of these elements are also high at Montréal-Duncan/Décarie. This is partially because this site has the highest median PM_{10} level. However, at Montréal-Duncan/Décarie, levels of Fe, Br, Mn, Cr are clearly enhanced, relative to PM_{10} and relative to the other Montréal site. The relatively high median Br and Pb concentrations are consistent with the influence of motor vehicles. Leaded fuel was in use during the early part of the monitoring program and consequently Pb levels have dropped considerably from 1984-86 to 1992. The higher median concentrations of Br and Fe could also be due to the traffic near the site. Although Mn, as MMT (methylcyclopentadienyl-manganese tricarbonyl), is used in Canada as a gasoline additive, the relatively high concentration at Montréal-Duncan/Décarie is likely a result of a nearby ferro and silico-manganese plant. This is also supported by the drop in Mn levels since the closure of this plant (Loranger and Zayed, 1994).

The effect of being located near the stack of an oil-burning boiler is evident in the median concentrations of V and Ni at Halifax, which are higher than at any other site. The high mean annual concentrations are due to a dramatic wintertime increase in the levels of V and Ni since Halifax concentrations are not much different than the other sites during the summer (not shown). However, some of the elevated Ni and V concentrations observed at Halifax are also likely due to use of oil for heating and electricity generation throughout the Maritime region and the east coast of the US. The link between Se and coal combustion is evident in the concentrations shown in Table 5.25. Median Se concentrations are higher in Montréal and Toronto, which are closest to the US Midwest and southern Ontario, where coal consumption is high.

As expected, Cl^- concentrations are higher at the coastal sites. A marine source of Br may also be affecting Vancouver and Victoria, but this does not show up at Halifax. Toronto and Montréal, Canada's two major eastern cities, have a higher median NO_3^- concentration than their western counterpart of Vancouver. This could be a reflection of the temperature dependence of NH_4NO_3 formation since the eastern cities have colder climates (Section 2.7.5 describes how particle nitrate formation is favoured by low temperatures). However, the higher NO_3^- concentration could also be due to higher levels of HNO_3 in Toronto and Montréal. Brook (1994) found that in the summer of 1993 the HNO_3 concentrations were about a factor or two higher in southern Ontario compared to the Vancouver area.

Figure 5.19 Comparison of Urban and Rural Concentrations (µg/m³) of the Most Abundant Species (Summer 1993).



As described in Brook (1997) the fraction of fine and coarse mass accounted for by soil (crustal material), $SO_4^{2-}/NO_3^-/NH_4^+$, NaCl and other elements were determined using mass-reconstruction techniques and the inorganic species data. This methodology does not account for carbonaceous species (organic and elemental carbon) which are known to contribute significantly to the total mass of atmospheric particulate matter. It is assumed that the difference between reconstructed mass and total mass indicates an upper limit for particle mass associated with organic and elemental carbon. The results for summer and winter for the NAPS sites are illustrated in Figure 5.20. Consistent with other studies (Malm et al., 1994; Countess et al., 1980) soil is the largest identifiable component in the coarse fraction, while $SO_4^{2-}/NO_3^-/NH_4^+$ dominates the identifiable components within the fine fraction. Figure 5.20 show the percentage of mass accounted for by each of these components as well as the percentages attributable to NaCl, other inorganic species and the percent of mass that is unexplained. On average, the $SO_4^{2-}/NO_3^-/NH_4^+$ component accounts for from about 20 to 45% of the fine fraction, depending upon site. The fraction of mass associated with the $SO_4^{2-}/NO_3^-/NH_4^+$ component is smallest at the three Prairie sites.

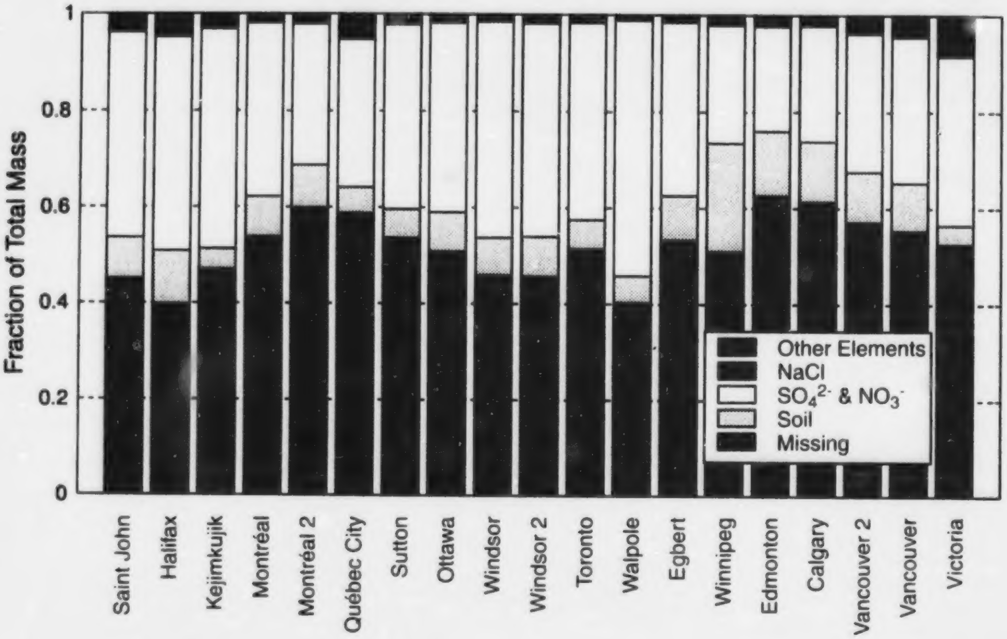
Soil-based inorganic compounds generally account for 5-15% of the fine mass, and are most significant to the overall mass in Winnipeg and least important in Toronto. There is very little seasonal difference in the percent of fine mass explained by soil. As expected, the NaCl component of the fine particles is larger at the sites near salt water. However, even at these locations it only accounts for a maximum of 5% of the $PM_{2.5}$. A large portion of the fine mass is not explained by the elements or compounds for which chemical analysis results are available. The average percent of unexplained mass is shown by the lower portion of the columns in Figure 5.20. The unexplained mass ranges from 40% at Halifax during both seasons to 70% of the total at the Vancouver sites during the winter. On average, only about 45% of the total mass can be explained by the mass reconstruction.

Table 5.25 Median Concentrations (μg/m³) of the Most Abundant PM_{10} Species for Selected Urban Sites (All Data 1986-1993).

Compound	Halifax	Montréal	Montréal Duncan	Ottawa	Toronto	Winnipeg	Calgary	Edmonton	Vancouver West 10th	Vancouver	Victoria
Sulphate	4.59	3.45	3.68	2.98	3.62	1.59	1.29	1.15	1.93	1.93	1.81
Nitrate	0.35	0.62	0.63	0.44	0.65	0.44	0.37	0.33	0.55	0.57	0.42
Sulphur	1.82	1.38	1.50	1.25	1.48	0.63	0.50	0.46	0.82	0.87	0.67
Aluminum	1.147	0.619	1.895	0.928	0.300	1.060	0.454	1.236	1.700	1.352	0.196
Calcium	0.113	0.856	1.079	0.618	0.800	1.013	0.986	0.441	0.167	0.204	0.125
Silicon	0.563	0.931	1.249	0.653	0.827	1.360	1.859	2.486	0.644	0.660	0.427
Chlorine	0.424	0.078	0.170	0.062	0.101	0.068	0.093	0.077	0.190	0.249	0.353
Iron	0.176	0.315	0.608	0.184	0.339	0.255	0.345	0.497	0.325	0.221	0.157
Potassium	0.094	0.179	0.232	0.138	0.147	0.164	0.170	0.172	0.118	0.139	0.091
Magnesium	0.064	0.071	0.122	0.089	0.109	0.350	0.140	0.100	0.110	0.198	0.084
Lead	0.015	0.042	0.070	0.018	0.046	0.019	0.041	0.057	0.058	0.055	0.054
Titanium	0.015	0.033	0.052	0.019	0.026	0.022	0.029	0.040	0.032	0.019	0.018
Zinc	0.019	0.046	0.068	0.020	0.042	0.017	0.018	0.018	0.024	0.029	0.015
Bromine	0.005	0.007	0.015	0.004	0.007	0.004	0.007	0.011	0.017	0.012	0.015
Phosphorus	0.027	0.034	0.036	0.022	0.026	0.012	0.013	0.012	0.015	0.019	0.010
Manganese	0.010	0.026	0.044	0.015	0.023	0.014	0.019	0.026	0.022	0.024	0.015
Barium	< DL	0.010	0.027	< DL	0.010	0.009	0.011	0.012	0.008	0.008	0.005
Scandium	0.005	0.015	0.018	0.011	0.014	0.015	0.014	0.009	0.008	0.005	0.004
Vanadium	0.098	0.008	0.013	< DL	< DL	< DL	0.002	0.004	0.005	0.006	0.006
Copper	0.008	0.010	0.015	0.011	0.011	0.006	0.004	0.006	0.014	0.014	0.002
Antimony	0.007	0.007	0.010	0.005	0.008	0.006	0.007	0.007	0.008	0.008	0.009
Tin	0.014	0.009	0.012	0.006	0.010	0.006	0.007	0.006	0.008	0.007	0.008
Nickel	0.020	0.003	0.004	0.001	0.002	< DL	< DL	< DL	0.003	0.002	0.003
Chromium	0.001	0.003	0.006	< DL	0.003	0.002	< DL	0.003	0.004	0.003	< DL
Selenium	< DL	0.001	0.001	< DL	0.001	< DL	< DL	< DL	< DL	< DL	< DL
Sampling Days	**251**	**536**	**266**	**412**	**545**	**411**	**445**	**426**	**503**	**362**	**368**

Figure 5.20 Mean Fractional Contributions to the Total Fine and Coarse Mass from the Chemical Species Attributed to Soil, $SO_4^{2-}/NO_3^-/NH_4^+$, NaCl and other Elements.

a) Fine – Summer



b) Coarse – Summer

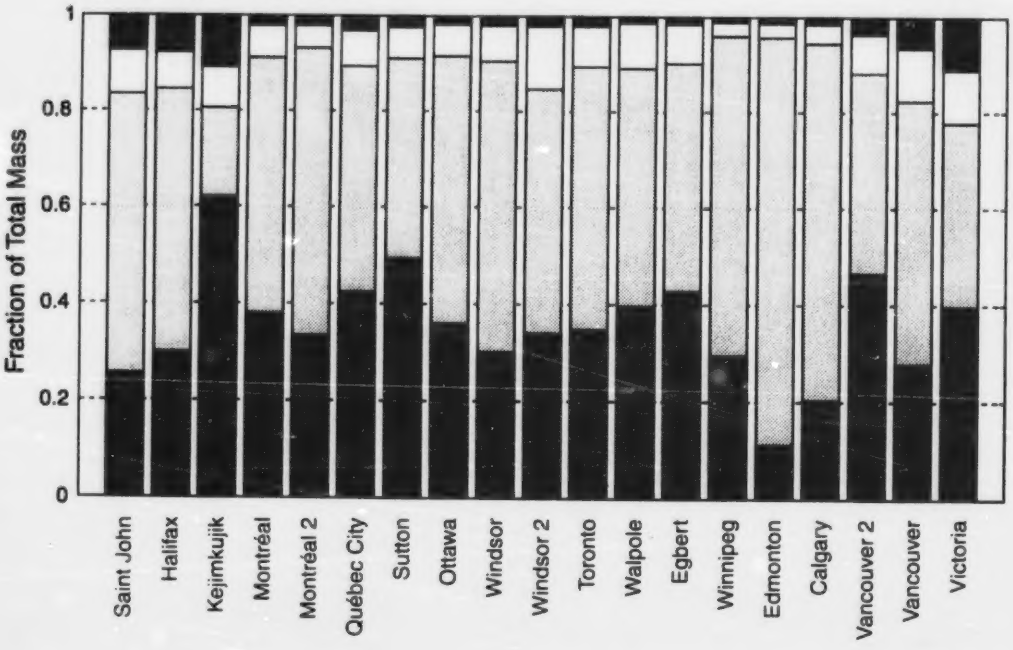
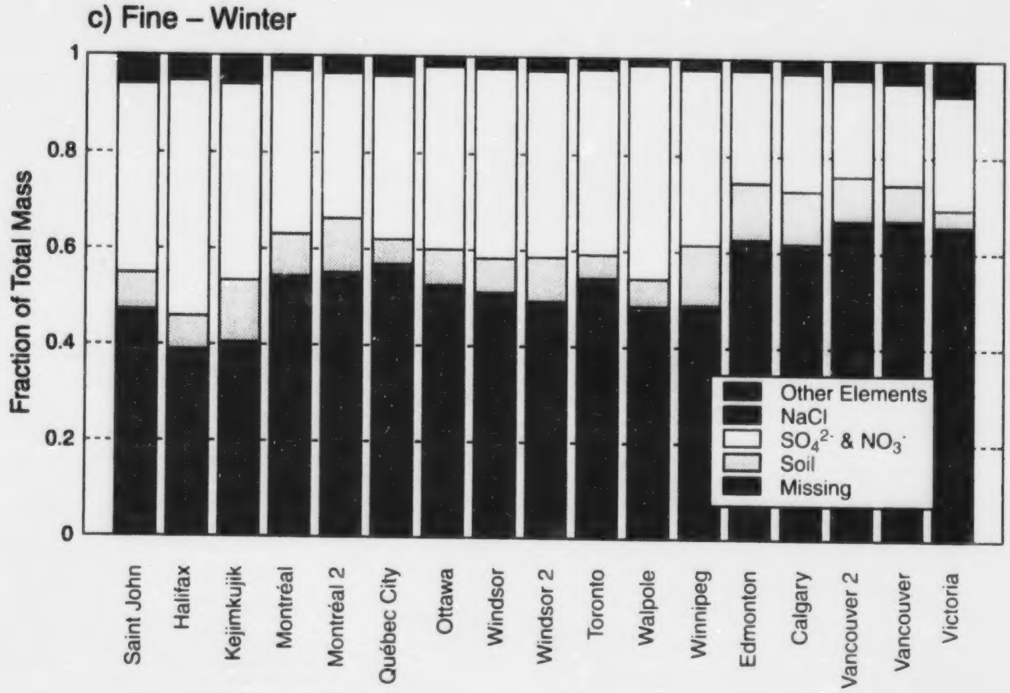
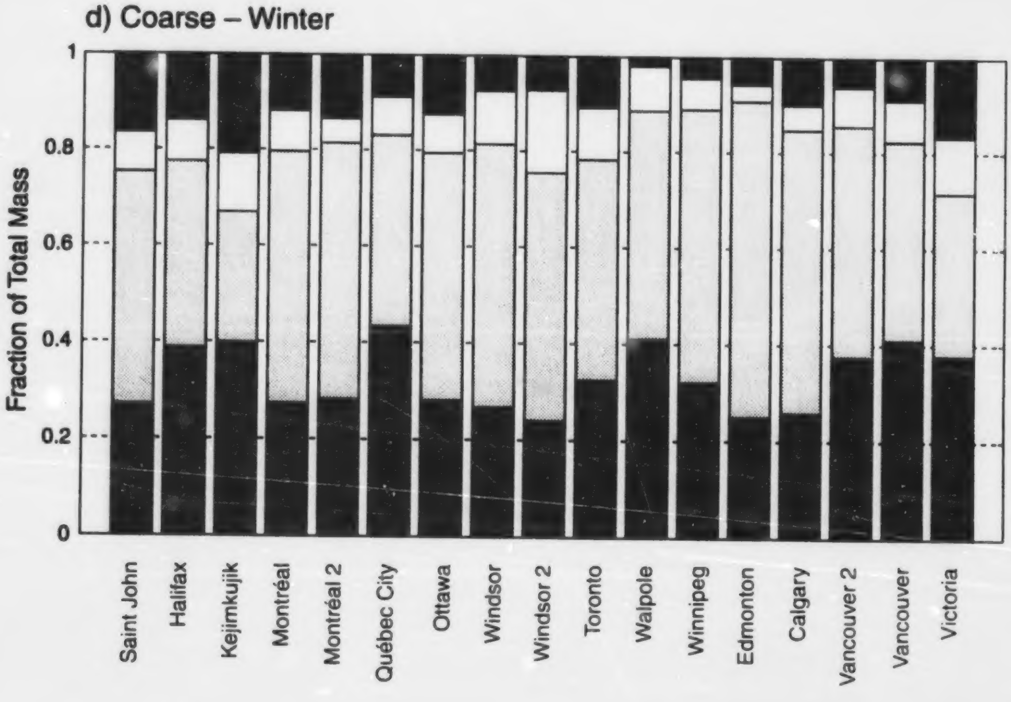


Figure 5.20 Mean Fractional Contributions to the Total Fine and Coarse Mass from the Chemical Species Attributed to Soil, $SO_4^{2-}/NO_3^-/NH_4^+$, NaCl and other Elements. *(continued)*


c) Fine – Winter


d) Coarse – Winter

As expected, the amount of coarse mass associated with the soil component is much larger than with fine particles. On average across all sites, the soil fraction is about 50%. However, there is considerable variability with values ranging from 21% at Kejimkujik to 75% at Edmonton. From the fine to the coarse particles, the $SO_4^{2-}/NO_3^-/NH_4^+$ component decreases substantially in importance and the NaCl fraction increases. During the winter, the coarse mass contribution by NaCl is larger at all sites. This is associated with use of road salt. At the coastal sites, this increase may also be due to increases in wind speed and hence, sea spray. The largest unexplained coarse fraction is observed at Kejimkujik. This could be associated with the forest environment throughout the region surrounding the site. This environment, plus the lack of agriculture and the proximity to the ocean may be the reason for the small percentage of soil-related elements. The forests around Kejimkujik could also be a source of organic material, leading to a relatively large unexplained mass fraction. The soil fractions are also relatively small at the other coastal sites, particularly Victoria. The inland sites with the smallest soil fractions are Québec City and Sutton, both of which are in regions with significant amounts of forested landscape. Figure 5.20 shows that 20-40% of the coarse fraction also remains unexplained.

As discussed, the unexplained mass is assumed to be associated with carbonaceous species. Under this assumption, fine particle carbonaceous species account for from 65% to 40% of the total mass, depending upon the site. There is apparently a greater percentage of carbon-related fine particles in the west (~62%) than there is on the east coast (40-46%). For 13 of the 19 sites, the estimated carbon percentage associated with the coarse particles is smaller than for the fine particles. Coarse particle carbonaceous species account for approximately 20-58% of total mass, depending on the site (excluding Kejimkujik and Edmonton). Slightly lower percentages occur in the wintertime, decreasing to about 40% of total mass.

5.4.4 Summary

On average across all sites, mean $PM_{2.5}$ accounted for 50% of mean PM_{10} and PM_{10} accounted for 50% of the TSP. The strength and nature of the correlations between TSP, PM_{10}, $PM_{2.5}$ and coarse particles varied depending upon site, and particle size. For example, the mean $PM_{2.5}$ to PM_{10} ratio ranged from ≤ 0.40 at three different urban sites in the Prairies to ≥ 0.60 at the rural and west coast sites. This ratio also varied substantially from measurement to measurement, but at most sites a majority (>50%) of the ratios were within ±10% of the median value.

At a majority of the sites, the daily variability in fine particle mass had a stronger influence on the variations in PM_{10} than did the coarse particle mass. This was most evident at the rural locations and at sites not heavily impacted by urbanization (i.e., traffic and construction). While this is consistent with the idea that fine particles control the variability in PM_{10}, it was not true at all sites or over all geographical areas. For example, the coarse mass dominated the PM_{10} variability at the Prairie sites and at the site most heavily impacted by traffic. Comparison of the urban and rural sites that were in relatively close proximity to one another indicated that particle mass concentrations were lower at the rural locations. This urban enhancement in concentration was greatest for the coarse particles. For example, the mean coarse particle concentration is 46% higher in Toronto compared to Egbert, while the mean amount of fine particles is only 28% higher in Toronto. This is also reflected in the urban versus rural correlation coefficients, which were systematically higher for fine particles and even higher for ambient SO_4^{2-}. The decrease in correlation from SO_4^{2-} to $PM_{2.5}$ suggests that some of the other chemical species contributing to the fine particle mass had more localized sources, which did not impact evenly on their neighbouring urban or rural sites.

Estimates of the amount of fine and coarse particle mass due to carbonaceous material were assumed using a mass balance approach. Depending upon site, only about 37 to 61 percent of the $PM_{2.5}$ could be explained given the measured concentrations of several inorganic ions and elements. Thus, carbonaceous material, which was likely to have been predominantly organic in nature, was responsible for about half of the overall fine particle mass. This fraction was higher in Alberta and British Columbia (~65%) than it was on the east coast (40-45%). Due to the increased importance of crustal material, a greater portion of the coarse particle mass (~70%) was explained by the inorganic constituents.

6 HUMAN EXPOSURE ASSESSMENT

The likelihood of an adverse response to particles is influenced by the degree of exposure, defined as any contact between the chemical at a specified concentration and the outer (e.g., skin) or inner (e.g., respiratory tract epithelium) surface of the human body (Sexton and Ryan, 1988; Lioy, 1990). Exposure implies the simultaneous occurrence of these two events in time and space (Ott, 1982). Changes in the degree of exposure are influenced by the duration (how long you are exposed), magnitude (concentration) and frequency (how often you are exposed) of exposure. Inhalation is the only exposure pathway considered in the assessment. This chapter will briefly: review the factors influencing exposure and the methods of determining air pollution exposure, review monitoring studies of personal and microenvironmental particle concentrations, present the results of a probabilistic exposure model, and discuss the relationship between current ambient levels of particles and human exposure in Canada.

6.1 CONCEPTS

Ambient particles are typically collected over a 24 h sampling period, although hourly average measurements of particle mass help illustrate the diurnal, weekly and seasonal variability in particle concentrations. Until more hourly data become available, expressing exposure over 24 h facilitates comparison of individual and population exposures with ambient particle levels.

Over a 24 h period, a person spends their time in many locations or microenvironments. US residents spend on average 21½ hours indoors, one hour outdoors, and 1½ hours in a vehicle. The data vary by age, gender and day of the week (US EPA, 1995b). For an individual, microenvironmental exposures lasting minutes to hours can be summed over one day and expressed by the following equation:

$$E_l = \sum_{j=1}^{J} c_{ij} t_{ij} \qquad (6.1)$$

where E_i equals the exposure to individual I, t_{ij} denotes the time the I-th individual spent in the j-th microenvironment, and c_{ij} denotes the average pollutant concentration for the I-th individual in the j-th microenvironment (Ott, 1982; Duan, 1991). This equation is based upon the concept that concentrations of pollutants measured in locations where people are, multiplied by the time spent in each place, will approximate personal exposure (Lioy, 1990). Therefore, individual and population exposures will vary, depending on the time spent in various microenvironments (frequency and duration of exposure) and the particle concentrations in those microenvironments (magnitude of exposure).

With people spending, on average, less than 10% of their time outdoors (Özkaynak et al., 1995; U.S. EPA, 1995b), it is important to understand what influences the concentrations of particles in other microenvironments.

6.2 MICROENVIRONMENTAL PARTICLE SOURCES AND CONCENTRATIONS

Microenvironmental levels of particles are a function of: indoor sources, outdoor particle levels, the fraction of ambient air penetrating indoors, filtration, air-exchange rates, decay rates, and resuspension rates (Clayton et al., 1993, Wallace et al., 1993, Thatcher and Layton, 1995; US EPA, 1982, 1996). For a microenvironment with no indoor sources of particles, the concentration can be illustrated by the following equation:

$$\frac{C_{in}}{C_{out}} = \frac{Pa}{a+k} \qquad (6.2)$$

where *p* is the penetration factor (a unitless measure of the ability of particles to penetrate the building envelope), *a* is the air exchange rate per hour (ach, or h^{-1}), and *k* is the particle decay rate (h^{-1}, a measurement of particle settling due to diffusion or sedimentation).

6.2.1 Sources

Indoor particles (Table 6.1) are attributed to six major source types: plant, animal, mineral, combustion, home/personal care and radioactive (Owen et al.,

Table 6.1 Sources of Indoor Particles (based on Owen et al., 1992)

Source Type	Description
plant	pollens, spores, molds, miscellaneous byproducts (finely ground grains, coffee, cornstarch)
animal	bacteria, viruses, hair, insect parts and byproducts, epithelial cells (eg. dandruff)
mineral	asbestos, talc, man-made mineral fibres, elemental particles (carbon)
combustion	tobacco smoke, cooking, heating appliances
home/personal care products	sprays, humidifiers
radioactive	radon progeny

1992). Plant materials are mostly of outdoor origin; pollens and spores settle out quickly but can be resuspended by vacuuming, sweeping or dusting. Animal and mineral particles have indoor and outdoor sources.

6.2.2 Penetration, Deposition and Resuspension

Penetration of ambient particles indoors is affected by several factors, including the physical and chemical characteristics of the particles and the mechanisms of home air exchange. Outdoor air enters a building through doors, windows and cracks, and as make-up air for heating and ventilating systems. Air-conditioned and energy efficient homes tend towards very low air exchange rates, while older homes are more "leaky". Air exchange rates can range from a minimum 0.1 air changes per hour up to 10 changes per hour when doors and windows are open. The Exposure Factors Handbook (US EPA, 1995b) presents arithmetic and geometric mean (SD) estimates of air exchange rates for the United States (0.63 [0.65] and 0.46 [2.25] each respectively).

Several early studies indicated penetration is more effective for fine particles than coarse particles. Penetration factors of approximately 0.6-0.7 have been reported for $PM_{2.5}$, with the coarser fraction (2.5 - 10 μm) probably penetrating less effectively (Colome et al., 1992; Dockery and Spengler, 1981a; Koutrakis et al., 1992; Lioy et al., 1990; and Yocom, 1982). Dockery and Spengler (1981a) reported that the net effect of full air conditioning was to reduce penetration factors from approximately 0.7 to approximately 0.3. The results of the pTEAM study, however (Özkaynak et al., 1993) show that the penetration factor (mean, lower and upper 95% confidence levels), calculated using a nonlinear statistical approach, for both fine and coarse particles is very close to unity (1.00; [0.89; 1.11] and 1.00; [0.85; 1.15], respectively). The authors expect that penetration rates would be lower outside of Riverside because of tighter house construction. The results of Thatcher and Layton (1995) agree with the pTEAM results, with experimentally derived penetrations factors for both fine and coarse particles not significantly different from one (i.e., full penetration of outdoor air indoors). Despite these recent experimental results, current scientific thinking (US EPA, 1996) concludes that the penetration factor for submicron particles is greater than for $PM_{2.5}$ which is greater than for coarse particles.

Once indoors, ambient particles settle out quickly by gravity or electrostatic forces. Average decay rates, due to diffusion or sedimentation, were calculated as part of the pTEAM study (Özkaynak et al., 1993) for $PM_{2.5}$ ($0.39 \pm 0.16\ h^{-1}$), PM_{10} ($0.65 \pm 0.28\ h^{-1}$), and the coarse fraction ($1.01 \pm 0.43\ h^{-1}$). The decay rate of sulphate ($0.16\ h^{-1}$), an indicator of the fine fraction, was less than that of $PM_{2.5}$. Thatcher and Layton (1995) calculated similar deposition velocities, with particles of size range 1 to 5 μm depositing at a rate of $0.46\ h^{-1}$, and particles of size range 5 to 10 μm depositing at a rate of $1.36\ h^{-1}$. Particles of size range 0.1 to 1 μm have negligible settling velocities, while particles >10 μm normally settle out of the air.

Once deposited, resuspension of particles readily occurs in homes and buildings with occupants as a result of indoor activities. Thatcher and Layton (1995) studied resuspension and showed that particles greater than 5 μm are easily resuspended, while smaller particles (< 1 μm) are "not" resuspended. Particles ranging from 1 – 5 μm can be resuspended with vigorous activity.

6.2.3 Concentrations

Three large-scale studies conducted in the United States as well as multiple small-scale studies have measured indoor levels of particles in homes. Each study followed slightly different methodologies, using monitors with different cutpoints and sampling for variable periods, thereby precluding direct comparison between studies. Thus, the results of several of these studies are presented (Table 6.2) for information purposes, and to further the subsequent discussion of individual and population exposure estimates.

Harvard's Air Pollution Health Effects Study, also called the Six Cities Study, investigated the respiratory effects of air pollutants in six cities in the United States: Portage, Wisconsin; Topeka, Kansas; Kingston/Harriman, Tennessee; Watertown, Massachusetts; St. Louis, Missouri; and Steubenville, Ohio (Spengler et al., 1981). During the first phase of this study, which began in 1979, indoor and outdoor air was sampled every six days in eight to eleven homes in each city. Respirable particle measurements were made using a cyclone sampler with a 3.5 µm cutpoint operating at a flow rate of 1.7 L/min. The results from this large-scale study show that mean indoor concentrations were greater than outdoor levels (Table 6.2) (Dockery and Spengler, 1981a; Sexton et al., 1984; Spengler et al., 1985).

Similar results were reported from the New York State Energy Research and Development Authority [ERDA] Study (Sheldon et al., 1989), which examined the impact of four combustion products on indoor particle levels. Week-long PM$_{2.5}$ samples were collected in two indoor locations in 394 homes and outdoors in a subset of these homes using the Harvard impactor. The samplers were equipped with two oiled impactor plates, designed to minimize the collection of particles larger than 2.5 µm. Mean indoor concentrations were approximately double the outdoor levels.

A large-scale field monitoring pTEAM study was undertaken in Riverside, California, in the fall of 1990 (Pellizzari et al., 1992; Özkaynak et al., 1993; Wallace et al., 1993; US EPA, 1996). The main goal of pTEAM was to estimate the frequency distribution of exposures to PM$_{10}$ for all non-smoking residents of Riverside (n = 139 000 ± 16 000) aged 10 and above. A pilot study was undertaken in Azusa, CA to test the sampling equipment (Wallace, 1996). A sample of 178 residents, selected using a probability-based design, participated by wearing a personal exposure monitor (PEM) for two consecutive 12 h periods (nominally 7 p.m. to 7 a.m. and 7 a.m. to 7 p.m.). The PEM consisted of a using a low-flow (4 L/min) impactor characterized by sharp cutpoints at 2.5 and 10.7 µm. Particles were collected on 37 mm Teflon filters mounted behind a greased impaction plate. The same monitor was modified to collect indoor and outdoor samples (stationary indoor monitors [SIMs] and stationary ambient monitors [SAMs], respectively) at participants' homes. Participants completed time-activity diaries and separately administered questionnaires about potential exposure to particle sources. The complete study design and sampling methods for the measurement of particles and subsequent elemental analyses are discussed elsewhere (Pellizzari et al., 1992; Özkaynak et al., 1993; Thomas et al., 1993).

Results from both the pilot study and the pTEAM study showed lower mean 24 h indoor concentrations of both PM$_{10}$ and PM$_{2.5}$ when compared with outdoor levels (Pellizzari et al., 1992; Wallace, 1996). Similar results were reported from some small-scale studies ([Bahadori et al., 1995; Diemel et al., 1981; Janssen et al., 1995; Kulmala et al., 1987; cited in Wallace, 1996] Colome et al., 1992; Lioy et al., 1990; Morandi et al., 1988) while other studies reported higher mean indoor concentrations. However, in many cases, the range of particle concentrations indoors and outdoors are similar.

Table 6.2 Indoor Concentrations of Particles (µg/m^3)

Reference	Location	Size fraction	Time	N	Indoor mean (range)	Outdoor mean (range)
Dockery & Spengler, 1981a	Portage, WI Topeka, KS Watertown, MA Kingston/Harriman, TN St. Louis, MO Steubenville, OH	$PM_{3.5}$ $PM_{2.5}$	annual 24 h (q 6 day) mean indoors annual mean outdoor	68	20 23 31 36 47 39	13 13 24 20 36
Sexton et al., 1984	Waterbury, VT	$PM_{3.5}$	24 h (8-8), every other day for two weeks	24	25 (6-69)	19 (6-30)
Spengler et al., 1985	Kingston/Harriman, TN	$PM_{2.5}$ out $PM_{3.5}$ in	24 h	101	42	18
Sheldon et al., 1989	Onondaga, NY Suffolk, NY	$PM_{2.5}$	collected during alt 15 min periods over 7 days, winter	433	36.7 (0.72-172) 46.4 (2.18-284)	16.8 (6.32-28.4) 21.8 (12-106)
[Wiener, 1988; Spengler et al., 1989; Özkaynak et al., 1990; Wiener et al., 1990][a]	Azusa, CA	PM_{10} $PM_{2.5}$	24 h (two 12 h periods)	9	58.7 (3.4)[b] 36.3 (2.6)	62.2 (3.5) 42.6 (3.0)
Pellizzari et al., 1992	Riverside, CA	PM_{10} $PM_{2.5}$	24 h (two 12 h periods)	178	79 43	91 50
[Diemel et al., 1981]	Arnhem, the Netherlands	< 3-4 µm, cutpoint not specified	24 h	101	140 (20-570)	64 (53.7-73.3)
[Kulmala et al., 1987]	Helsinki, Finland	fine (< 1 µm) coarse (> 1 µm)		100	16 (4-67)[c] 13 (3-63)	20 (5-82) 16 (3-91)
[Janssen et al., 1995]	Amsterdam	PM_{10}	24 h, home		35 (19-65)	42 (32-51)
[Bahadori et al., 1995]	Nashville, TN	$PM_{2.5}$ PM_{10}	12 h daytime 12 h nighttime 12 h daytime 12 h nighttime	10	22 15 16 12	33 32 23 22
Lioy et al., 1990	Phillipsburg, NJ	PM_{10}	24 h	14	54 (25-60)	60 (12-165)
Colome et al., 1992	Orange County, CA	PM_{10}	24 h	8	32.7 (2.2)[a]	51.5 (4.9)

[a] bracketed studies cited in Wallace, 1996; [b] Mean (SE); [c] geometric mean (95% range).

6.2.4 Source Apportionment

Additional data analyses from many of the studies estimated source emission rates and the contributions of various sources to microenvironmental particle concentrations (Tables 6.3, 6.4, 6.5, and 6.6). The data from these tables are discussed in turn below.

Spengler et al., (1981) categorized the Harvard Six Cities data by number of smokers in the home and concluded cigarette smoke was the major source of indoor particles in smoking homes (Table 6.3). Similar results were presented from separate analysis of the Kingston/Harriman data (Letz et al., 1984; Spengler et al., 1985) and the Watertown data (Lebret et al., 1987). Dockery and Spengler (1981a) applied a simple conservation of mass model, to the phase one data set of the Six Cities Study, to determine contributions from three sources: cigarette smoking, heating and cooking. Processes affecting indoor levels were limited to infiltration, air-exchange rates, pollutant removal or decay and indoor sources. The impact of smoking one pack of cigarettes a day was significant, raising 24 h indoor respirable particle levels by approximately 18 μg/m^3, compared to an additional 15 μg/m^3 by other sources. In fully air-conditioned buildings, where infiltration rates are greatly reduced, smoking was estimated to contribute an additional 42 μg/m^3 of respirable particles. Smoking and gas stoves were also identified as possible sources of sulphates.

The second phase of the Harvard Six Cities Study took place between 1983 and 1988 (Neas et al., 1994). It involved 1,237 Caucasian never-smoking children, aged 7 to 11 years, which were drawn from a cohort of 6,273 children from the six cities. Indoor PM$_{2.5}$ levels in 200 to 300 homes selected from each city were measured over two consecutive one-week sampling periods during the summer and winter using the Harvard impactor. Sampling was continuous except for an 8 h period each weekday in which the child was at school. Neas et al. found that annual household average PM$_{2.5}$ concentrations were 31.2 μg/m^3 higher for the 580 children living in consistently smoking households than for the 470 children living in consistently non-smoking households. Based on the indoor measurements and questionnaire responses, they reported that the estimated number of cigarette packs smoked per day was a good indicator of annual average indoor PM$_{2.5}$ concentrations. A concentration of 30 μg/m^3 corresponded to the impact of smoking approximately one pack of cigarettes per day. Analysis of a subset of the phase two data in three communities (Spengler et al., 1987) also revealed mean PM$_{2.5}$ concentrations in homes with smokers about 30 μg/m^3 greater than homes without smokers.

Santanam et al. (1990) reported on a subset of the Harvard Six Cities Study data collected between 1986 and 1987 in Steubenville and Portage. Particle samples were analyzed for 25 elements using XRF spectrometry. Concentrations of selected tracer elements were analyzed by principal component analysis (PCA) to apportion source contributions. Based on these analyses, tobacco smoke was identified as the largest source of indoor PM$_{2.5}$ in smoking households in both cities (Table 6.4). Sulphur-related sources accounted for 8 to 9 μg/m^3 in Steubenville during the summer, but contributions during the winter were not detected. In Portage, sulphur-related sources accounted for 5 to 6 μg/m^3 during the summer and winter. Woodsmoke accounted for approximately 4 μg/m^3 in Steubenville and only 1 μg/m^3 in Portage during the winter. Auto-related sources accounted for 4 μg/m^3 in Steubenville during the summer and 2 to 5 μg/m^3 in Portage during the winter and summer. Unexplained source contributions range from 2 to 12 μg/m^3 in Steubenville and 1 to 6 μg/m^3 in Portage. The sizable unexplained portion was attributed to the fact that since some tracer elements from distinct sources covaried, the PCA method could not distinguish between the associated source types.

Analysis of the ERDA study data from Suffolk and Onondaga Counties, New York, (Sheldon et al., 1989; Leaderer and Hammond, 1991) demonstrated that smoking significantly increased indoor PM$_{2.5}$ concentrations. Koutrakis et al. (1992) furthered the analysis, looking at the contributions of the combustion sources to indoor PM$_{2.5}$ levels. Samples were analyzed by X-ray fluorescence for 16 elements. A simple physical model was applied to determine the contribution of indoor and outdoor sources. Gas stoves were not found to contribute to indoor PM$_{2.5}$ levels during this study. In homes containing only wood-burning or kerosene stoves, or none of the identified sources, approximately 60-70% of the indoor PM$_{2.5}$ levels were attributed to outdoor sources. However, in homes occupied by cigarette smokers, smoking accounted for 54% of PM$_{2.5}$ levels, and outdoor sources and other indoor sources an additional 30 and 16%, respectively. Large home-to-home variations in PM$_{2.5}$ levels were observed.

Table 6.3 Indoor Concentrations of Particles as a Function of Smoking ($\mu g/m^3$)

Reference	Location	Size fraction	Indoor		Outdoor
			classification	mean level	
Spengler et al., 1981	six cities	PM$_{3.5}$ Annual (q6d) 24 h mean	no smokers one smoker two or more smokers	24.4 (n = 35) 36.5 (n = 15) 70.4 (n = 5)	21.1 (n = 55)
Neas et al., 1994	six cities	PM$_{2.5}$, 2 cons. weeks, summer & winter	no smokers smokers	17.3 (n = 470) 48.5 (n = 580)	
Spengler et al., 1985	Kingston/ Harriman TN	PM$_{3.5}$	no smokers smokers	28 (n = 73) 74 (n = 28)	18
Lebret et al., 1987	Watertown, MA	PM$_{2.5}$ 24 h	no smokers smokers	21.6 (n = 70/74) 54 (n = 147/152)	
Santanam et al., 1990	Steubenville Portage	PM$_{2.5}$ (4 pm to 8 am) one week in summer and winter	no smokers smokers no smokers smokers	19.5 (w)[a] 24.5 (s)[b] 43.6 (w) 49.9 (s) 14.8 (w) 13.6 (s) 34.6 (w) 24.9 (s)	18.7(w) 29.5(s) 10.3(w) 13.6(s)
[Jenkins et al., 1994, 1995][g]	12 cities	PM$_{3.5}$	non smoking offices restricted smoking unrestricted smoking	18 (13)[c], n = 629 28 (16), n = 297 58 (33), n = 113	
[Lebret et al., 1990]	Ede & Rotterdam, the Netherlands	RSP (cutpoint not specified) week-long, winter	no smokers smokers	30 (geom. mean) 70	45
[Heavner et al., 1995]	New Jersey & Pennsylvania	PM$_{3.5}$ 14 h home samples 7 h workplace samples	home – no smokers home – smokers workplace – no smokers workplace – smokers	27.6 (19.9)[d] n = 58 86.7 (145.4) n = 30 30.3 (17.6) n = 52 67.0 (44.3) n = 28	
[Quackenboss et al., 1989]	Tucson, Arizona	PM$_{10}$ PM$_{2.5}$	no smokers < one pack/day > one pack/day no smokers < one pack/day > one pack/day	30.3 (19.9)[d] n = 43 46.2 (29.1) n = 27 75.0 (57.2) n = 18 15.2 (15.5) n = 45 27.3 (23.6) n = 26 60.8 (50.8) n = 17	
[Quackenboss et al., 1991]	Tucson, Arizona	PM$_{2.5}$, spring/summer/fall PM$_{2.5}$, winter	no smokers smokers no smokers smokers	10[e] 20 13 (n = 26) 36 (n = 24)	
[Kim & Stock, 1986]	Houston area	PM$_{2.5}$, week long day and night 12 h samples	no smokers smokers	10.8 (4.9)[d] n = 6 33.0 (4.7) n = 5	12.0 (5.9) 24.7 (7.4)
[Morandi et al., 1986]	Houston	PM$_{3.5}$ (approximate) week long daytime (12 h) samples	no smokers smokers smokers with AC smokers without AC	19 89 114 52	
[Chan et al., 1995]	Taiwan	PM$_5$, 24 h, summer winter	one smoker (11.1 butts) one smoker (6.1 butts)	44 (32)[d] 107 (44)	27 (15) 92 (40)

[a] (w) = winter; [b] (s) = summer; [c] mean (median); [d] mean (standard deviation); [e] median levels; [f] arithmetic mean (geometric mean); [g] bracketed studies cited in Wallace, 1996

Table 6.4 Contribution of Smoking to 24 h Indoor Particle Concentrations ($\mu g/m^3$)

Reference	Size fraction	Contribution	Comments
Dockery & Spengler, 1981[a]	$PM_{3.5}$ (24 h average)[a]	0.88 1.23	homes without air conditioning homes with air conditioning
Letz et al., 1984	$PM_{3.5}$	29.4 (n = 57, 24 h)	ETS component
Lebret et al., 1987	$PM_{2.5}$ (24 h average)[a]	0.8	
Santanam et al., 1990	$PM_{2.5}$ (16 h averages)	20-27 10-25	Steubenville Portage
Leaderer and Hammond, 1991	$PM_{2.5}$ (24 h average)[a]	2.1 (1.9 - 2.3)	
Koutrakis et al., 1992	$PM_{2.5}$	26 (54%)	ETS component in smoking homes
Wallace, 1996	PM_{10} $PM_{2.5}$	29-37 27-32	
Özkaynak et al., 1993	PM_{10} (24 h averages) $PM_{2.5}$	2 1.5	
[Lebret et al., 1990] cited in Wallace, 1996	RSP (24 h averages) cutpoint not specified	2 - 5 10	one cigarette one cigar
[Chan et al., 1995] cited in Wallace, 1996	PM_5 (24 h average)	16	ETS-related component

[a] increase in the daily concentration per cigarette

Koutrakis et al. (1992) also looked at the sources of various elements in their analysis. The elements Mn, Se and Ni were associated mostly with outdoor sources. The elements K, Ca and Cu were associated with indoor sources. In particular, smoking, consumer products and dust resuspension were cited as possible sources of chlorine. Smoking, wood-burning and kerosene heaters were associated with K. Possible sources of Ca included dust resuspension, biological particles and particles formed during showering. Finally, vacuum cleaners, kitchen ventilation, fans and kerosene heaters were associated with Cu. Cadmium was exclusively associated with outdoor sources in non-source and wood-burning homes. In homes with kerosene heaters or smokers, outdoor contributions of Cd dropped to 10-40%, suggesting that at least one of these two sources emits Cd. Sulphur was also largely associated with outdoor sources in non-source and wood-burning homes. However, kerosene heaters were also found to be a large source of indoor S, contributing 40-50%, while smoking contributed approximately 10%. Silicon was associated with outdoor sources and kerosene heaters, wood-burning and other indoor sources.

Source contributions were estimated from the pTEAM results using a non-linear method of solving a mass-balance model. Averaged over homes containing no known indoor sources (n = 244), outdoor sources accounted for 76% of indoor $PM_{2.5}$ and 66% of PM_{10} in Riverside (Figure 6.1). Unidentified indoor sources accounted for 14% of $PM_{2.5}$ levels and 26% of PM_{10} levels. Smoking and cooking, the two main identified indoor sources, accounted for only 4-5% each in both size fractions. In homes where these activities took place, each accounted for approximately 20-30% of the indoor particle concentrations (Figure 6.2 and 6.3). Other household activities appeared to make smaller contributions to indoor particle levels.

The pTEAM results showed that tobacco smoke significantly increased indoor PM_{10} and $PM_{2.5}$ concentrations (Özkaynak et al., 1993; Wallace, 1996). Overnight indoor levels for both PM_{10} and $PM_{2.5}$ in smoking homes, as well as personal PM_{10} levels, were about twice as high as corresponding levels in non-smoking homes. Averaged over smoking households (n = 40), outdoor air still remained the largest source of indoor $PM_{2.5}$, accounting for 60% compared to 25-30% by smoking and 7-16% by other unidentified sources.

Figure 6.1 Sources of Fine Particles ($PM_{2.5}$) (left) and Thoracic Particles (PM_{10}) (right) in All Homes (Riverside, CA). Source: Özkaynak et al., (1995b)

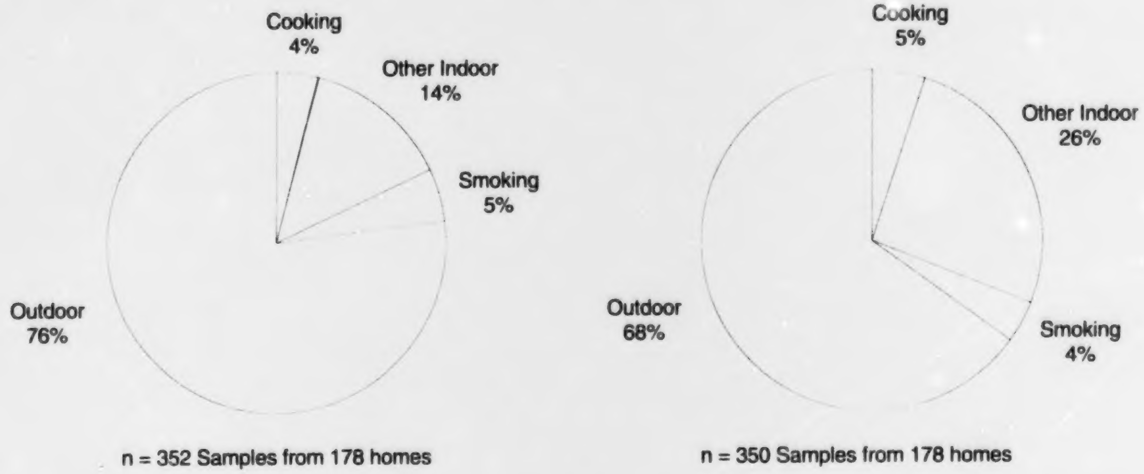


Figure 6.2 Sources of Fine Particles ($PM_{2.5}$) (left) and Thoracic Particles (PM_{10}) (right) in Homes with Smokers (Riverside, CA). Source: Özkaynak et al., (1995b)

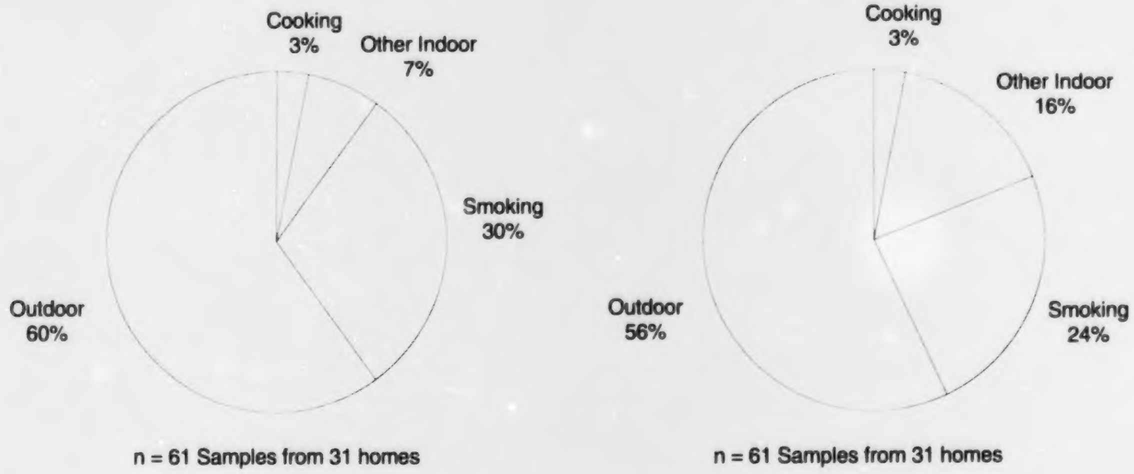
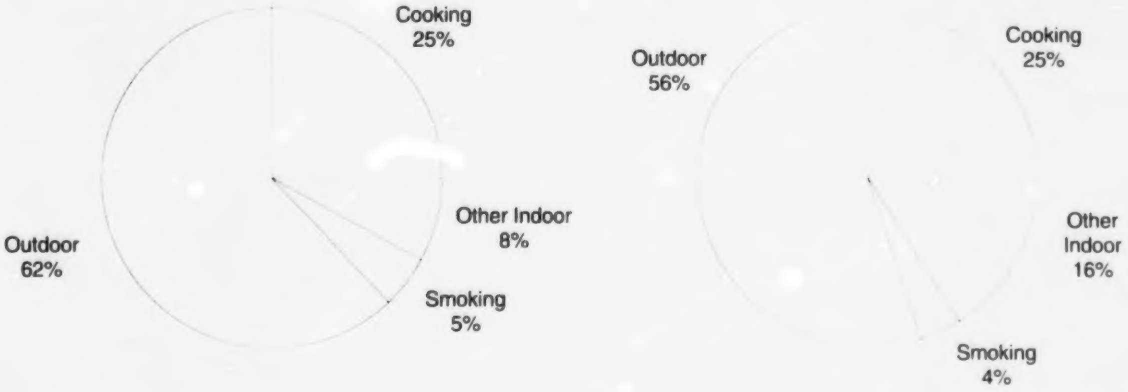


Figure 6.3 Sources of Fine Particles ($PM_{2.5}$) and Thoracic Particles (PM_{10}), left and right panels, Respectively, for Homes with Cooking During Data Collection (Riverside, CA). Source: Özkaynak et al., (1995b)



n = 62 Samples from 33 homes

n = 62 Samples from 33 homes

Using elemental fingerprinting and applying multivariate analysis methods, Özkaynak et al. (1995b) demonstrated that the contribution to 12 h average indoor PM_{10} and $PM_{2.5}$ of each cigarette smoked is about 2 and 1.5 µg/m^3, respectively. Cooking added 13 µg/m^3 to daytime $PM_{2.5}$ levels and increased daytime PM_{10} levels by about 6 µg/m^3 per minute of cooking. The elements Al, Ti, P, S and Zn were mainly associated with outdoor sources, while the elements Cu, Ca and Cl were mainly associated with unidentified indoor sources. Smoking accounted for 16-21% of the elements Ca, K and Cl. Outdoor air provided 70-100% of the indoor concentrations for most elements.

Several smaller studies also reported indoor levels of particles according to smoking prevalence (Chen et al., 1995; [Heavner et al., 1995; Jenkins et al., 1994. 1995; Kim and Stock, 1986; Lebret et al., 1990; Morandi et al., 1986; Quackenboss et al., 1989, 1991; cited in Wallace, 1996]) (Table 6.3). The data clearly show smoking increases the levels of both PM_{10} and $PM_{2.5}$. Results from instantaneous sampling, performed as part of a study in the Netherlands (Lebret et al., 1990; cited in Wallace, 1996), indicate that the indoor concentration of fine particles varies temporally depending upon the time since (or during) smoking (Table 6.5).

Indoor particle measurements were made in three middle-class, non-smoking homes in Chapel Hill, North Carolina during November and December of 1987 (Kamens et al., 1991). Particle samples were collected using two dichotomous samplers ($PM_{2.5}$ and PM_{10}), and 37 and 47 mm prototype personal sampling units, all co-located. Sampling periods consisted of at least two 8 h daytime periods and three 13 h evening-to-early morning periods. Particle size distributions were measured using an electrical mobility aerosol analyzer (EAA) and two laser optical aerosol counters. Particle morphology was analyzed using scanning electron microscopy (SEM). Measurements from all three homes showed that 37% of the estimated total mass of the collected material was in the fine fraction, 26% in the coarse fraction, and 37% greater than 10 µm. These size distributions varied considerably. For example, EAA measurements showed that during periods of cooking, particles less than 0.10 µm in diameter contributed 30% of the total EAA particle volume. Based on EAA data and SEM images, soot and sulphate particles associated with cooking appeared to be the largest contributors to the fine fraction. Morandi et al. (1986) also reported significantly higher respirable particle levels associated with cooking compared to homes where no cooking occurred. Vacuuming and sweeping were the most significant indoor sources of large particles (>10 µm), while biological and mineral-based particles were most closely associated with the coarse fraction (Kamens et al., 1991).

Table 6.5 Influence of Smoking on Instantaneous RSP Concentrations

Time Since Smoking	N	RSP[a] (geometric mean, µg/m^3)
No smoking	98	41
More than one hour ago	18	52
Between ½ and 1 hour ago	7	76
Less than ½ hour ago	27	141
During the measurements	54	191

[a] cutpoint not specified

source: Lebret et al., 1990; cited in Wallace, 1996

The impact of kerosene heaters on indoor air quality was studied by Mumford et al. (1991). Measurements were made in eight mobile homes in Apex, North Carolina. Selection criteria included heater type, regular heater usage, trailer size of 150-225 m^3, use of electric stoves or heating systems so that emissions from wood and gas stoves could be ignored, and no cigarette smokers. The sulphur content of the kerosene fuel was 0.024%. Measurements were made 6.5 hours per day, 3 days per week, and were restricted to evenings only. The indoor samples were collected on Teflon-coated glass fibre filters using a 10 µm inlet separator. Outdoor samples were collected using a high-volume sampler. The results of the study showed that, on average, the kerosene heaters did not affect indoor PM_{10} levels. However, in 2 of the 8 homes, significant effects were observed.

The impact of woodstoves on indoor particle levels was investigated by Sexton et al. (1984). As part of the Harvard wood-burning study, indoor levels of respirable particles were measured in a total of 19 wood-burning and 5 non-wood-burning homes in Waterbury, Vermont during the winter of 1981/1982. All occupants were nonsmokers. Indoor and outdoor particle levels were measured using Bendix cyclone preseparators attached to Harvard/EPRI portable sampling pumps. Sampling took place over 24 hours every other day for two-week periods. Indoor particle levels were significantly higher than outdoor levels, with an average indoor/outdoor ratio of 1.6. However, no statistical difference was found between 24 h average particle levels in wood-burning and non-wood-burning homes. A linear regression model that incorporates data on air-exchange rates, house volume, fuel use and outdoor levels accounted for only about 20% of the variance of indoor particle levels, suggesting large home-to-home differences in indoor particle levels.

Traynor et al. (1987) estimated the emissions of TSP from four different wood-burning stoves: 3 airtight and 1 non-airtight. TSP was collected on 47 mm Teflon filters. Source strengths were calculated by assuming that the indoor air was well-mixed, and that steady-state conditions applied. The airtight stoves were found to emit little TSP (2.5-8.7 mg/h) relative to the non-airtight stove (26-230 mg/h). Background emission rates were estimated to be 1.1-1.6 mg/h. Different operating styles were suggested as an explanation for the large variation in TSP emission rates estimated for non-airtight stoves.

The ambient data from Highsmith et al. (1988a; cited in Wallace, 1996), which increased by about 50% at night, suggest an influence of wood-burning on ambient levels. In this study, indoor levels of fine particles were greater in homes with wood-burning stoves compared to homes without (Table 6.6) in contrast with the data from Sexton et. al. Subsequently, Highsmith et al., (1988b; cited in Wallace, 1996) studied the effect of different types of portable humidifiers supplied with tap or distilled water on indoor particle concentrations in one home in Boise, Idaho. The study showed that the use of tap water in the humidifier had a dramatic effect on indoor particle levels compared to the use of distilled water (Table 6.6).

6.2.5 Conclusions

Indoor concentrations of PM_{10} and $PM_{2.5}$ are significantly influenced by ambient concentrations and air exchange rates. The data in Table 6.3 show that indoor levels can be higher than outdoor levels or lower than outdoor levels. Wallace (1996) notes that in areas where the outdoor concentrations are fairly high, indoor concentrations are less, whereas indoor concentrations greatly exceed outdoor concentrations in areas where the outdoor levels are relatively low. Clearly, a source of indoor particles is ambient air that has penetrated indoors.

Recent studies indicate both PM_{10} and $PM_{2.5}$ can readily penetrate into buildings (penetrations factors close to unity). Wallace (1996) calculated the fraction of outdoor fine and coarse particles found in homes at equilibrium for a range of air exchange rates (Figure 6.4) based upon the pTEAM estimates for penetration rates and deposition. The fraction of ambient PM_{10} indoors will lie somewhere between

Table 6.6 Influence of Indoors Sources on Indoor Concentrations

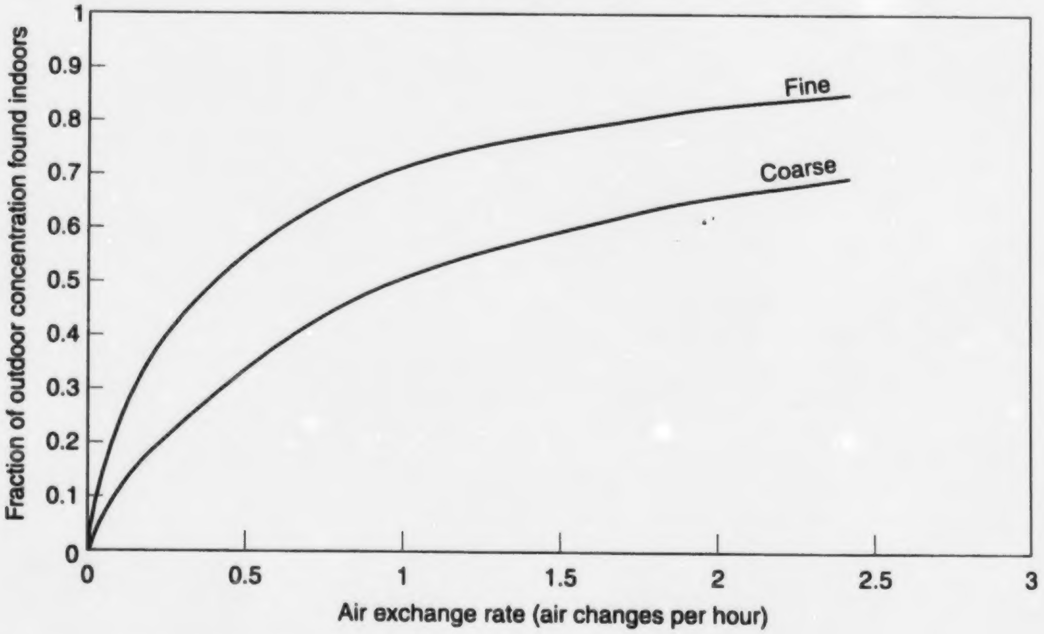
Reference	Sample	Particle size	Indoor levels
[Highsmith et al., 1988[a], 1991]*	10 homes with wood-burning stoves 10 homes without	$PM_{2.5}$, eight consecutive 12 h periods coarse particles, cutpoint not specified	26.3 18.2 10.2 9.7
[Morandi et al., 1986]	13 homes, with cooking without cooking	$PM_{3.5}$ (approximately)	27 20
[Highsmith et al., 1988b]	one home, background level; with ultrasonic humidifier & tap water; ultrasonic humidifier & distilled water	$PM_{2.5}$, 6-8 h samples	11 593 27

* [studies] cited in Wallace, 1996

the fine and coarse particle curves. In Canada, where building construction emphasizes energy efficiency and, therefore, low air-exchange rates, the fractions of fine and coarse particles of ambient origin that will be found indoors under equilibrium will tend toward 50% or less, particularly in the winter.

Once inside, the larger particles tend to settle out more quickly than smaller particles, however, the larger particles are easily resuspended as a result of indoor activities. Of the indoor sources of particles, in smoking homes, cigarette smoking has been identified as the most significant in the three large-scale studies conducted in the United States, and in many of the small-scale studies. Cooking was also consistently identified as a source of indoor particle levels, both PM_{10} and $PM_{2.5}$. In contrast, kerosene heaters and wood-burning stoves (perhaps surprisingly) were not found to consistently raise indoor particle levels. Unknown indoor sources remain, contributing approximately 25% to both PM_{10} and $PM_{2.5}$ levels.

Figure 6.4 Fraction of Indoor Particulate Matter (PM) from Outdoor Airborne PM, Under Equilibrium Conditions, as a Function of Air-exchange Rate, for Two Different Size Fractions. Source: US EPA, (1996)



Deposition rate = 0.39/h for fine particles (<2.5 µm), 1.01/h for coarse (>2.5 µm and <10 µm)

6.3 DIRECT MEASUREMENT OF PERSONAL EXPOSURE

There are three types of personal exposure: (1) Personal exposure to ambient particles, both ambient particles outdoors and ambient particles that have infiltrated indoors, (2) Personal exposure to non-ambient particles, (i.e., generated indoors), and (3) Personal exposure to total PM (ambient plus non-ambient). Exposure to total PM can be accurately measured if individual study participants carry personal monitors throughout their normal daily activities. With people spending 90% of their time indoors, total personal exposure (3 above), should theoretically be similar to indoor levels.

6.3.1 Personal Exposure Studies

As part of the Six Cities Study, Spengler et al. (1985) measured personal respirable particles (<3.5 µm), in addition to indoor and outdoor levels, in Kingston/Harriman, Tennessee. Ninety-seven volunteers participated during the 46-day sampling period in the winter of 1981. Twenty-four-hour indoor concentrations averaged 42 ± 2.6 (SE) µg/m^3 (n = 266); personal exposures averaged 44 ± 2.8 (SE) µg/m^3 (n = 249). Ambient concentrations measured at a central site were approximately 25 µg/m^3 (n = 71) less than mean personal and indoor concentrations.

Personal (n = 189), indoor (n = 101) and outdoor (n = 54) PM$_{10}$ levels were monitored for two weeks in the winter of 1988 in Phillipsburg, New Jersey (Lioy et al., 1990). The eight participating homes of 14 non-smoking, non-smoke-exposed individuals were within 2 km of a grey iron pipe manufacturing company. Twenty-four-hour average outdoor PM$_{10}$ concentrations ranged from 12 to 165 µg/m^3 at three rooftop sites (Table 6.7). Mean 24 h indoor concentrations ranged from 25 to 60 µg/m^3. Personal exposure levels averaged over 24 h were higher than outdoor concentrations and quite variable, ranging from 40 to 130 µg/m^3.

The pTEAM pilot study reported mean (SE) 24 h personal exposures approximately twice as high as indoor and outdoor for both PM$_{10}$ (112 vs 58.7 vs 62.6 µg/m^3) and PM$_{2.5}$ (70 vs 36.3 vs 42.6 µg/m^3) (Wallace, 1996). Twenty-four-hour personal PM$_{10}$ concentrations measured in the main study were also higher than all other monitored concentrations, ranging from 34 to 287 µg/m^3 (Table 6.7). However, the positive bias measured in the personal and microenvironmental samplers should be considered when evaluating the study results; measured PM$_{10}$ concentrations may be overestimated (Clayton et al., 1993; Thomas et al., 1993).

Population-weighted daytime PM$_{10}$ personal exposures (median 129.7 µg/m^3) were about 50% higher than both indoor (81.7 µg/m^3) and outdoor (84.1 µg/m^3) levels (Table 6.8). Daytime personal PM$_{10}$ exposure was about twice as high as the nighttime personal exposure (median 66.2 µg/m^3). The latter was slightly less than nighttime SAM concentrations (median 74 µg/m^3) and slightly higher than nighttime SIM concentrations (median 52 µg/m^3) (Pellizzari et al., 1992).

Table 6.7 Distributions of 24 hour PM$_{10}$ Concentrations

Monitor	n	24 hour PM$_{10}$ concentration (µg/m^3)										
		Arithmetic mean	Standard error	Geometric mean	Standard error	Min.	Percentiles					Max.
							10	25	50	75	90	
data from Lioy et al., 1990												
SAM	54	60	–	48	–	12	–	–	–	–	–	165
SIM	101	54	–	42	–	25	–	–	–	–	–	60
PEM	189	86	–	66	–	40	–	–	–	–	–	130
modified from Pellizzari et al., 1992												
SAM	153	91.2	4.1	81.4	3.7	15	47.2	60.9	78.3	112.5	159.7	280.4
SIM	157	79.0	4.1	68.5	3.8	20	32.9	45.1	65.3	106.4	143.6	324.8
PEM	161	112.5	6.0	100.7	5.6	34	52.1	70.1	102.5	148.2	183.5	286.9

Personal PM_{10} exposures were different, although the ambient concentrations were similar, for 37 non-smoking adults compared with 45 children, presumably non-smokers (Janssen et al., 1995; cited in Wallace, 1996). Subjects were monitored for one day per week over 7-8 weeks. The mean wintertime 24 h personal exposures (range) were 61 (36-113) $\mu g/m^3$ for the adults compared to 104 (59-194) $\mu g/m^3$ for the children. Mean ambient concentrations were 42 (32-51) $\mu g/m^3$ and 38 (25-56) $\mu g/m^3$ respectively. Twenty-four hour indoor concentrations in the adult homes averaged 35 (19-65) $\mu g/m^3$.

Other studies also support the observation that personal exposure to fine particles averages higher than indoor and outdoor measurements (Dockery and Spengler, 1981b; Sexton et al., 1984; Morandi et al., 1988). Spengler et al. (1980) also reported that 12 h personal respirable samples averaged 2.5 times greater than outdoor levels.

In all these studies, personal exposure was measured to be higher than either the indoor or the outdoor PM measurements. The exceptions are studies in disabled or elderly persons (Bahadori et al., 1995; Tamura and Ando, 1994; cited in Wallace, 1996). In a study of patients with COPD, Bahadori et al., 1995; (cited in Wallace, 1996) monitored indoor and outdoor PM_{10} and $PM_{2.5}$ levels for 12 consecutive 12 h periods. The patients wore PEMs during the daytime, alternating between a PM_{10} and $PM_{2.5}$ nozzle. Preliminary analysis of the data, which is population specific, indicated personal exposures were similar to outdoor concentrations for both size fractions. Individual PEM values were 30 to 50% of the SAM values measured outside the subject's house (Tamura and Ando, 1994; cited in U.S. EPA, 1996).

6.3.2 "Personal Cloud"

In non-smoking households, daytime personal exposure levels are always higher than indoor and outdoor particle concentrations (Spengler et al., 1985; Özkaynak et al., 1993; Neas et al., 1994; Thatcher and Layton, 1994). Wallace et al. (1991) suggested that this finding – higher personal exposures – may be explained by a "personal cloud effect" in which a person's movements or activities contribute to increased personal exposures.

Table 6.8 shows that overnight personal PM_{10} levels are similar to both indoor and outdoor levels, while indoor levels of PM_{10} and $PM_{2.5}$ are similar to outdoor levels during the day and drop off at night. Time-activity data may help explain the nighttime similarities in personal and indoor levels; while sleeping for about two-thirds of the overnight monitoring period, participants were not engaged in particle generating or re-entraining activities (Clayton et al., 1993). Examination of daytime and nighttime differences in personal, indoor and outdoor particle concentrations in the pTEAM study (Table 6.9) revealed a significantly lower PM_{10} exposure for working people than for participants who stayed home (Clayton et al., 1993; Özkaynak et al., 1993; Wallace et al., 1993). While at home, personal activities such as cooking, cleaning the house and smoking were related to higher personal exposures. Re-entrainment of house dust as a result of activities such as sitting on chairs and sofas or walking across carpets also increased personal exposure (Thatcher and Layton, 1995; Wallace et al., 1993).

An alternative explanation for the personal cloud could be attributable to the PEM over sampling particles larger than 10 µm. PM_{10} concentrations measured with the personal and microenvironmental monitors were consistently higher than the SAM measurements. Additional factors that may influence the differences in personal exposure and indoor and outdoor particles concentrations include: the presence of strong indoor sources, air-exchange rates, occupational exposures, the spatial variability of ambient particles and commuting habits.

As noted above, smoking is a major source of increased particle concentrations. Its contribution to the "personal cloud" is evident primarily at night (Table 6.9). Repace and Lowery (1980) estimated that the amount of particulate matter directly inhaled by the average smoker is 2-3 orders of magnitude greater than the concentration of environmental tobacco smoke (ETS) measured with PEMs.

The results of Bahadori et al. (1995; cited in Wallace, 1996) indicate that the "personal cloud" is made up mostly of coarse particles ($PM_{2.5}$ -PM_{10}). The results of Thatcher and Layton (1995), which indicated coarse particles are easily resuspended compared with fine particles, tend to support those results. The pTEAM results showed a 50-100% increase in 14 of 15 elements found in the indoor aerosol in the personal cloud. This would imply that the "personal cloud" consists largely of particles resuspended by personal activity.

Table 6.8 Weighted Distributions of Personal, Indoor and Outdoor Particle Concentrations (modified from Pellizzari et al., 1992)

			Particle concentrations (µg/m³)												
				Percentiles											
		n	Min.	10	25	50	75	90	Max.	Mean	Standard error	Geom. Mean	Standard error	Standard deviation	Geom. standard deviation
$PM_{2.5}$ (day)	SAM	167	7.4	14.9	23.4	35.5	60.1	102.2	187.8	48.9	3.5	37.7	2.5	38.1	2.8
	SIM	173	2.8	11.5	19.3	33.5	61.5	101.0	238.3	48.2	4.1	35.0	3.3	41.3	2.6
$PM_{2.5}$ (night)	SAM	161	3.4	14.5	23.0	35.0	64.9	120.7	164.2	50.5	3.7	37.2	3.1	41.0	3.5
	SIM	166	2.9	10.0	14.8	25.9	48.9	82.7	133.3	36.2	2.2	26.7	1.9	29.9	3.1
PM_{10} (day)	SAM	165	16.2	42.8	56.9	84.1	110.8	157.2	406.6	94.9	5.5	82.7	4.1	59.6	3.1
	SIM	169	16.6	30.9	49.5	81.7	127.2	180.7	512.8	94.7	5.7	78.2	5.0	63.1	3.0
	PEM	171	35.1	59.9	86.1	129.7	189.1	263.1	454.8	149.8	9.2	128.7	8.5	88.5	3.3
PM_{10} (night)	SAM	162	13.6	39.3	53.6	74.1	103.7	167.8	222.9	86.3	4.4	74.5	4.0	51.3	3.6
	SIM	163	14.1	25.2	33.5	51.6	84.8	116.9	180.3	62.7	3.2	53.1	3.1	40.3	3.8
	PEM	168	19.1	36.6	48.1	66.2	98.8	135.0	278.3	76.8	3.5	67.9	3.1	42.4	3.0

Table 6.9 Effects of Particle-generating Activities on Mean PM_{10} Personal Exposures (Pellizzari et al., 1992)

	PM_{10} personal exposures ($\mu g/m^3$)									
	Daytime					Nighttime				
Activity	n	Mean	Standard error	Geom. mean	Standard error	n	Mean	Standard error	Geom. mean	Standard error
Work[a]	59	126.8	11.6	107.6	10.7					
No work	111	162.1*	9.9	141.6*	8.5					
Housework[b]	110	161.5*	11.0	142.2*	9.6	64	73.0	5.6	65.9	4.9
No housework	61	125.0	11.4	104.4	10.6	104	79.5	3.2	69.4	2.8
Spraying[c]	70	158.3	12.0	138.5	10.6	55	81.5	5.9	69.7	5.5
No spraying	101	143.1	10.5	121.6	8.9	113	74.6	4.1	67.1	3.5
Smoke[d]	61	155.2	15.3	131.4	15.9	29	104.2*	8.0	96.6*	6.7
No smoke	110	146.8	7.7	127.3	6.3	139	71.4	3.3	63.3	2.7
Exhaust[e]	31	96.5	8.7	85.5	7.0	7	—	—	—	—
No exhaust	121	166.3*	9.0	144.8*	8.1	140	77.5	3.8	68.2	3.1

* indicates that the means or geometric means for persons with and without the activity are significantly different; the asterisk is shown beside the higher member of the pair.

[a] *work*: identifies participants who have a paid job outside of the home and does not include self-employed in the home, full-time student, full-time homemaker, out of work just now but usually employed, retired or disabled, and other.

[b] *housework*: consisted of vacuuming, dusting, carpet cleaning, indoor cooking and using a clothes dryer.

[c] *spraying*: included either pump- or propellant-type spraying of paints, cleaners, disinfectants, air fresheners, hair care products, perfumes/colognes, deodorants, cooking products, lubricants, insecticides, repellents and plant care products.

[d] *smoke*: is one or more cigarettes, cigars or pipefuls.

[e] *exhaust*: identifies participants or other household members potentially exposed to exhaust from vehicles running in attached garages.

Analysis of the pTEAM data suggested that the "personal cloud" is not due to variations in sampling characteristics of the monitors, nor to organic particles from people and their clothes. The excess personal exposure is likely due to generation or re-entrainment of particles during personal activities (Clayton et al., 1993; Wallace et al., 1993). However, the exact nature of the "personal cloud" has not yet been determined.

6.4 SURROGATES OF PERSONAL EXPOSURE

6.4.1 Exposure Estimates Using FAM

Particle concentrations measured at centrally located fixed ambient monitors (FAMs) in epidemiological studies are indicators of the extent of the contribution of ambient particles to personal exposure. This assumes that, ignoring microscale fluctuations in particle concentrations, population exposure to ambient PM can be represented by the 24 h average ambient concentrations measured at the central site (FAM). On this basis, 24 h average population exposures across Canada would range from 15-42 $\mu g/m^3$, the mean PM_{10} concentrations averaged from 1984-1995 at urban sites in the ambient monitoring network (Table 6.10). Similarly, mean 24 h $PM_{2.5}$ exposure would range from 8.5-20.2 $\mu g/m^3$, based upon FAM measurements at urban central sites over the same timeframe. Median and high end (90th percentile) 24 h FAM measurements representing personal ambient exposures are also presented.

Table 6.10 Estimated 24 h Population Exposure Based Upon Central Site Fixed Ambient Monitoring Data (range, µg/m^3)

	mean	median	high end exposure (90%)
PM_{10}	15-42	8-36	19-84
$PM_{2.5}$	8.5-20.2	4.8-17	10.9-39

data from dichot samplers, 1984-1995

However, as described in Chapter five, there are wide variations in daily measurements of PM at FAMs due in part to the location of the monitor, meteorology, intra-city differences, and diurnal patterns. In addition, it has been previously established that concentrations measured at FAMs do not correlate well with readings obtained simultaneously with personal monitors (Binder et al., 1976; Cortese and Spengler, 1976; Wallace, 1979; Ott and Flachsbart, 1982; U.S. EPA, 1982a). Exposure to non-ambient particles is not included in the personal exposure as represented by the FAM.

Table 6.11 summarizes the recent studies that measured personal exposure to total PM, including some additional exposure studies explained in detail elsewhere (US EPA, 1996). Almost all of the studies were conducted on urban and suburban residents. Personal exposures were usually greater than either the indoor (SIM) or outdoor (SAM) particle concentrations. Nine studies confirm the poor correlation between personal exposures and ambient particle concentrations, as evidenced by the R^2 values shown in the table. Of the remaining studies, the R^2 values range from 0.04 to 0.70, many of them significantlly different from zero. Some of the individual studies are discussed in the following paragraphs.

Correlational analysis between 24 h SAM, SIM and PEM pTEAM measurements show outdoor levels weakly correlated ($r_s = 0.41$) with personal PM_{10} exposures (Clayton et al., 1993; Pellizzari et al., 1992). Daytime correlations between personal and outdoor concentrations measured at the residence (SAM) (0.35) and at the fixed site (FAM) (0.37) suggest that outdoor monitors do not adequately represent personal exposure to total PM_{10}, particularly during the day. Correlations of personal and residential concentrations with fixed-site concentrations showed higher nighttime correlations between fixed-site and SAM data (Table 6.12). Correlations of indoor data (SIMs) with fixed-site data (FAMs) were lower; correlations with personal data were lower still (Clayton et al., 1993). The authors concluded that measurements of ambient air would be unable to predict personal exposure to total PM_{10} for most persons accurately.

In Kingston and Harriman (Spengler et al., 1985), correlation coefficients between personal exposure and ambient concentrations were low and not statistically significant ($r = 0.07$, $p = 0.30$); the correlation between indoor and personal concentrations was much stronger ($r = 0.7$, $p = 0.0001$). Cross-sectional (daily) regressions calculated from the Lioy et al., 1990 data (Wallace, 1996) also reported low personal-outdoor correlation coefficients; the median R^2 (range) was 0.06 (0.00 to 0.39). Unique results from Tokyo show personal PM_{10} exposures averaging well below outdoor concentrations, with a correlation coefficient of 0.68, likely because of minimal generation and resuspension of particles indoors (Tamura and Ando, 1994; cited in Wallace, 1996).

Most of the studies report poor cross-sectional personal-outdoor correlations. If there is a lot of variability in particle concentrations from sources poorly correlated with centrally located FAMs, than the percent of variance that can be explained by the FAM data is small. Sampling error, a non-random sample, very strong indoor sources and personal activities all contribute to a poor correlation between FAMs and PEMs. FAMs measure ambient particles and PEMs measure personal exposure to ambient and non-ambient PM.

Table 6.11 Comparison of Personal Exposure Concentrations with Simultaneous Ambient Concentrations

Location	Year	PM (μm)	n	time	mean PEM ($\mu g/m^3$)	mean SAM ($\mu g/m^3$)	R^2 PEM vs SAM	p	Reference
Ansonia	1973	5	20	24 h	115	59	NS	NS	Binder et al., 1976
Watertown	1975	3.5	18	24 h	35	17	0	NS	Dockery & Spengler, 1981
Steubenville	1976	3.5	19	12 h	57	64	0.19	NR	Dockery & Spengler, 1981
Topeka	1979	3.5	46	12 h	30	13	0.04	NS	Spengler et al., 1980
Toronto	1981	25	13	8 h					WHO/UNEP, 1982
non-asthmatic	winter				122	68	0.15	NS	
non-asthmatic	summer				124	78	0.1	NS	
asthmatic	winter				91	54	0	NS	
asthmatic	summer				124	80	0.07	NS	
Kingston/Harriman	1981	3.5	97	12 h	44	18	0	NS	Spengler et al., 1985
Zagreb	1982	5	12	1 wk					WHO/UNEP, 1982
	summer				114	55	0	NS	
	winter				187	193	0.5	NR	
Waterbury	1982	3.5	48	24 h	36	17	0	NS	Sexton & Spengler, 1984
Bombay	1982	3.5	15	24 h				NR	WHO/UNEP, 1984
	winter				127	117	0.26	NR	
	summer				67	65	0.2	NS	
	monsoon				58	51	0.02		
Beijing	1985	3.5	20						WHO/UNEP, 1985
	winter			24 h	177	421	0.07	0.09	
	summer			1 wk	66	192	0.03	NS	
Houston	1988	3.5	30	12 h	27	16	0.34	<0.05	Morandi et al., 1988
Philipsburg	1988	10	14[a] 14[b]	24 h	86 76	60 60	0.04 0.25	0.008 0.001	Lioy et al., 1990
Azusa	1989	2.5 10	9 9	24 h	79 115	43 62	0.01 0.01	NS NS	Perritt et al., 1993
Riverside	1990	10	141	24 h	113	84	0.23	NR	Clayton et al., 1993
Tokyo	1992	10	7	48 h	37	56	0.68	0.000	Tamura et al., 1996
Nashville, TN	1995	2.5 10	10	12 h day	22 33	23 32	0.42 0.48	0.000 0.000	Bahadori et al.,

Note: Please refer to US EPA (1996) for complete reference information.

*n = number of individuals carrying personal monitors; Year = year study was performed.

NS = not statistically significant from 0; NR = p value not reported, but mentioned as significant

[a] = 14 subjects carried PEMs for 14 days for 191 valid measurements;

[b] = three outliers are removed and regression is for 188 measurements

Table 6.12 Correlations of PM_{10} and $PM_{2.5}$ Residential Data and Personal PM_{10} Concentrations with Fixed-Site Concentrations

Personal and residential data	Particle fraction	Daytime fixed-site	Nighttime fixed-site
Residential Outdoors (SAM)	2.5	0.83	0.96
SAM	10	0.66	0.93
Residential Indoors (SIM)	2.5	0.70	0.71
SIM	10	0.51	0.59
Personal (PEM)	10	0.37	0.54

compiled using data from Clayton et al., 1993

When individual (longitudinal) regressions of personal exposure with the nearest outdoor site are calculated, the correlations improve (R^2=0.46, range: 0.02-0.82) when compared with the cross-sectional correlations (Lioy et al., 1990). Similar improvements were noted when the longitudinal regressions of personal exposures and outdoor concentrations were calculated for the pTEAM pilot study data (Wallace, 1996) and in Tokyo (Tamura and Ando, 1994; cited in Wallace, 1996). Median individual longitudinal correlations for individual adults or children were also higher (R^2=0.25, range 0.17-0.85 and R^2=0.40, range 0.00-0.96, respectively) compared with cross-sectional personal-outdoor correlation coefficients for adults (R^2= 0.02) and children (R^2= 0.11) (Janssen et al., 1995, cited in Wallace, 1996). The improvement in correlation coefficients suggests that, for individuals who are not exposed to microenvironmental sources of particles, and whose day-to-day activities are fairly repetitive, ambient levels of particles directly reflect their exposure to particles originating in the ambient air.

The correlation between outdoor measurements at FAM or SAM and personal exposure measurements also increases when the mean of the PEM values from the personal exposure studies is calculated. Mage and Buckley (1995) conclude that the uncertainty in predicting mean community exposure to total PM is smaller than the uncertainty associated with predicting individual personal exposure to PM. They propose the following: when a large portion of the total particulate matter is composed of fine particles, and when coarse particles are relatively constant in a community, the correlation between PEMs and FAMs is higher, because the fine particles readily penetrate indoors. Thus, ambient fine particles measured at the centrally located FAM can serve as an indicator of community (population) exposure. However, personal exposure of smokers and individuals exposed to ETS is not represented by the FAM (Mage and Buckley, 1995; US EPA, 1996).

In summary, a 24 h time-weighted average exposure estimate using ambient data from a central site (FAM), may be able to represent a part of the distribution of personal exposure to particles (fine fraction more so than coarse fraction), particularly for non-smoking individuals who are not exposed to indoor particle sources. However, the 24 h time-weighted average exposure estimate using solely ambient data does omit potential "peak" exposure to non-ambient particles in microenvironments such as indoors at home, at work, in-transit, and other indoor locations (discussed in sections 6.2 and 6.3). Therefore, estimates of personal exposure based upon ambient data do not tell us anything about personal exposure to non-ambient particles; total personal exposure is underestimated.

6.4.2 Time-Weighted Average Exposure Estimates Using Ambient and Indoor Data

Introducing microenvironmental exposures into a time-weighted average exposure estimate will theoretically improve estimates of personal exposure to total PM. Results from the pTEAM study show 24 h PEM and SIM PM_{10} data are highly correlated (0.70). Nighttime correlations between personal and outdoor (SAM) and indoor (SIM) data (0.62 and 0.80, respectively) are uniformly high (Pellizzari et al., 1992; Clayton et al., 1993). Moderate correlations of personal exposures to indoor concentrations were reported for both size fractions by Bahadori et al.

(1995; cited in Wallace, 1996). The high correlation between personal exposure and indoor concentrations combined with the time spent indoors indicates that indoor microenvironments are the most important contributors to overall exposure.

However, like the indirect personal exposure estimates using solely FAMs, indirect exposure estimates (E_I) using indoor and ambient particle measurements will likely underestimate personal exposure. Spengler et al., (1985) estimated that only 54% of the variance in personal exposure could be predicted using the equation:

$$E_i = C_i T_i + C_o T_o \qquad (6.3)$$

where concentration and time are modelled for the indoor (I) and outdoor (o) environments. The additional variability could be because concentrations measured during personal exposure studies in unoccupied microenvironments do not include the increase in indoor particle concentrations generated by vacuuming, dusting, indoor cooking, carpet cleaning and using the clothes dryer. Similarly, ambient monitors do not include the increased particle levels generated during outdoor activities such as gardening, burning leaves/rubbish, outdoor cooking or recreational activities, nor vehicular emissions and resuspension of roadway dusts.

6.5 PREDICTED DISTRIBUTION OF PERSONAL EXPOSURES TO PM$_{10}$ IN CANADA

Using the findings from population-based field investigations, personal and population exposure models have been developed (Johnson et al., 1994; Özkaynak et al., 1996) that combine ambient measurements of pollutants with information on age-specific time-activity and estimates of microenvironmental pollutant concentrations. A Monte-Carlo based framework developed by Harvard researchers was applied to Canadian ambient PM$_{10}$ measurements, demographics, and smoking rates by region to generate personal exposure distributions (Özkaynak et al., 1995). The results from that modelling-based analysis are described in the subsequent sections. Predictions are limited to exposures occurring via inhalation.

6.5.1 Methodology

Model Structure

The PM$_{10}$ population exposure model estimates personal exposures on a daily (24 h average) period by summing the time-activity weighted microenvironmental exposures using equation 6-1:

$$E_i = \sum_{j=1}^{m} E_{ij} = \sum_{j=1}^{m} f_{ij} \cdot C_{ij}$$

where E_{ij} = exposure to individual I in microenvironment j (µg/m^3), f_{ij} = the fraction of time spent by person I in microenvironment j during the 24 h prediction period, C_{ij} = the average PM$_{10}$ concentration (µg/m^3) in microenvironment j when individual I is present and m is the number of microenvironments considered in the model.

These are the six different microenvironments included in the exposure model: 1) outdoors, 2) indoors at home during daytime (7:00 a.m.-7:00 p.m.), 3) indoors at home during nighttime (7:00 p.m.-7:00 a.m.), 4) in-transit, 5) indoors not home or bar/restaurant, 6) bar/restaurant. Exposures to PM$_{10}$ from smoking and cooking sources were also considered by combining the time during which individuals are exposed to either source, with predicted PM$_{10}$ concentrations from ETS or cooking, in environments where cooking or smoking occurs. The general structure of the physical model for indoor PM$_{10}$, developed initially for the pTEAM study, is:

$$C_{in} = \frac{P \cdot a}{a+k} C_{out} + \frac{N_{cig} S_{smk} + T_{cook} S_{cook}}{(a+k)Vt} + \frac{S_{other}}{(a+k)V} \qquad (6.4)$$

where C_{in} = PM$_{10}$ concentration indoors, C_{out} = PM$_{10}$ concentration outdoors, P = penetration fraction (unitless), a= air exchange rate (hr^{-1}), k= PM$_{10}$ decay or deposition rate (hr^{-1}), Ncig = number of cigarettes, V = house volume (m^3), T_{cook} = cooking time (hr), t = monitoring period (12 hrs), S_{smk} = estimated source strength for cigarette smoking (mg/cig), S_{cook} = estimated source strength for cooking (mg/min), and S_{other} = estimated source strength for other indoor sources (mg/hr).

Simulation Procedure

The model produces estimates of the distribution of 24 h average exposures by using input distributions that represent variability of microenvironmental concentrations, time-activity patterns and contact rates among the modelled individuals. Characterizations of uncertainty for the PM_{10} model were limited to parameter uncertainty; scenario and model uncertainty were assumed to be negligible. A two-dimensional Monte-Carlo simulation approach was used to propagate the estimated uncertainty about model inputs (parameter variability and uncertainty) through to the distributions of the prediction endpoints. The details of the simulation methodology and the uncertainty analysis can be found elsewhere (MacIntosh et al., 1995; Özkaynak et al., 1995a).

6.5.2 Data Bases and Input Distributions

The types of distributions used to represent inter-individual, spatial and temporal variability, and parameter uncertainty were based on physical data as much as possible and supplemented by the professional judgement of the contractors when required. Normal or lognormal distributions were assigned to distributions representing inter-individual differences in microenvironmental concentrations and exposure durations. For distributions representing knowledge uncertainty, uniform distributions were used to characterize inputs when the quantity and quality of data were low. When moderate amounts of data were available, triangular distributions were typically used; normal or lognormal distributions were used when substantial amounts of quality data were available.

Ambient Air

Available ambient PM_{10} measurements for 1990-1993 were obtained from Environment Canada. Table 6.13 lists the station numbers and sites used to develop ambient PM_{10} distributions for each of the five regions (British Columbia, the Prairies, Ontario, Québec, and the Maritimes) used for the analysis. Four sites (Sutton, Kejimkujik, Egbert & Walpole) were used to represent rural PM_{10} concentrations. Analysis of the data showed a lognormal fit for most of the data with some seasonal variations. Site specific geometric means (GM) and geometric standard deviations (GSD) were estimated and combined with demographic information to produce seasonal and regional GM and GSD estimates for the ambient PM_{10} distributions.

Table 6.13 List of Canadian PM_{10} Monitoring Sites Used in the Personal Exposure Model

Region	Station #	Site
Maritimes/ Atlantic	10101 40201 40203 40204 40205 30311 30101 30501	St. John's Saint John Saint John Saint John Saint John Sydney Halifax Keji
Québec	50104 50109 50307 54101	Montréal1 Montréal2 Québec City Sutton
Ontario	60104 60204 60211 60417 60424 61901 64401	Ottawa Windsor Windsor2 Toronto Toronto2 Walpole Island Egbert
Prairies	70119 80109 80110 80209 80211 90130 90204 90227	Winnipeg Regina Regina2 Saskatoon Saskatoon2 Edmonton Calgary Calgary2
British Columbia	100106 100111 100118 100109 100303	Vancouver Vancouver2 Vancouver1 Vancouver3 Victoria

Demographics

Demographic data (age, gender, and location, rural or non-rural dwellers) were obtained from the 1991 Census (Statistics Canada, 1993). The relative size of the population near each monitor was sorted by region and by urban versus rural classification. The population was divided into four age categories (10-19 years, 20-24 years, 25-64 years and 65-99 years) to match the available time-activity information.

Time-Activity Patterns

Time-weighted microenvironmental exposures were calculated using time-activity diaries from a random sample of about 3,000 US residents recently obtained during the National Human Activity Pattern Survey (cited in Özkaynak et al., 1995a). An analysis of the daily time-activity diaries to determine whether significant differences exist in time spent indoors and outdoors by geographic location and season revealed subjects living in the north US and near Canada spending slightly more time outdoors during the summer and less time outdoors in the winter than the rest of the US population. Because the minor seasonal differences in outdoor times were small, estimates of time spent each day in each of the six study microenvironments were calculated from the entire sample of time-activity diaries.

Smoking

Data obtained from Health Canada reports (cited in Özkaynak et al., 1995a) on the prevalence of cigarette smoking by age and gender for all of Canada, cigarette consumption rates by region (regardless of age) and age-specific US cigarette smoking rates published by the Department of Health and Human Services, (cited in Özkaynak et al., 1995a) were used to develop age-dependent smoking prevalence rates by region. Table 6.14 summarizes the percentage of smokers and Table 6.15 breaks down the smoking population, according to the number of cigarettes, by region, age and gender. In modelling ETS exposures, smokers were assumed to be exposed to ETS in the residential and in-transit microenvironments. Exposure to ETS for smokers in the remaining microenvironments and non-smokers in all microenvironments were simulated using data obtained from sources detailed in Özkaynak et al., 1995a.

Indoor PM_{10} Concentrations

Residential indoor concentrations are modelled by a semi-empirical physical model (discussed above) which assumes contributions to indoor PM_{10} from: outdoors, environmental tobacco smoke (ETS), cooking, and other unaccounted indoor sources. The data bases and input source strength estimates required to construct and set parameters for the indoor concentration models were obtained from the pTEAM study results (Özkaynak et al., 1995b, 1996).

PM_{10} concentrations inside automobile cabins were assumed to be similar to concentrations on the highway, which were predicted from a relationship developed between a highway monitor (Duncan Décarie site in Montréal) and the more centrally located site in the city. The highway monitor recorded on average, 20% higher PM_{10} concentrations than the average of the two monitors. ETS concentrations in automobile cabins were simulated based upon consideration of source strength and air exchange rates. Indoor PM_{10} concentrations in public and commercial buildings, and in restaurants and bars were based on survey data from the US and England.

Table 6.14 Smoking Population by Gender, Age & Region (%)

Age*	Gender	British Columbia	Prairies	Ontario	Quebec	Atlantic
14-19	M	24	26	21	34	28
	F	27	29	23	38	31
20-24	M	37	39	31	51	41
	F	31	33	26	43	35
25-64	M	31	33	26	43	35
	F	29	31	25	41	33
>65	M	15	16	13	21	17
	F	13	14	11	18	15

* For age under 13, smoking rate is assumed to be zero.
Source: Health Canada, 1994 (cited in Özkaynak et al., 1995a)

Table 6.15 Smoking Population by Number of Cigarettes (%)

Region	Gender	Number of Cigarettes Smoked per Day		
		1-10	11-25	>25
British Columbia	M	14.4	64.6	21
	F	34.4	62.2	
Atlantic	M	16.8	70.4	12.8
	F	33.6	59.2	7.3
Québec	M	18.3	36.6	18.1
	F	30.4	59.9	9.7
Ontario	M	26.5	62.9	10.6
	F	31.2	64.4	4.5
Prairies	M	23.4	69.6	7
	F	27.3	70	

Building Factors

Average house size was estimated using the Canadian census (living habits) data on the average number of rooms per household. Estimated Canadian home volumes were compared to the distribution of 1,836 US residence volumes reported by Pandian (cited in Özkaynak et al., 1995a). Since the distributions were found comparable, the more complete house volume data from the US database was used in modelling exposures. The distribution of house volumes was represented by a lognormal fit to the data.

Air exchange data were obtained from the Brookhaven Laboratory (BNL) data base. An analysis of the potential for air exchange rates to vary systematically by region (north, south, and other) revealed similar air exchange rates in the spring, fall and winter months for the North region. Regional differences were apparent for some seasons (summer, winter).

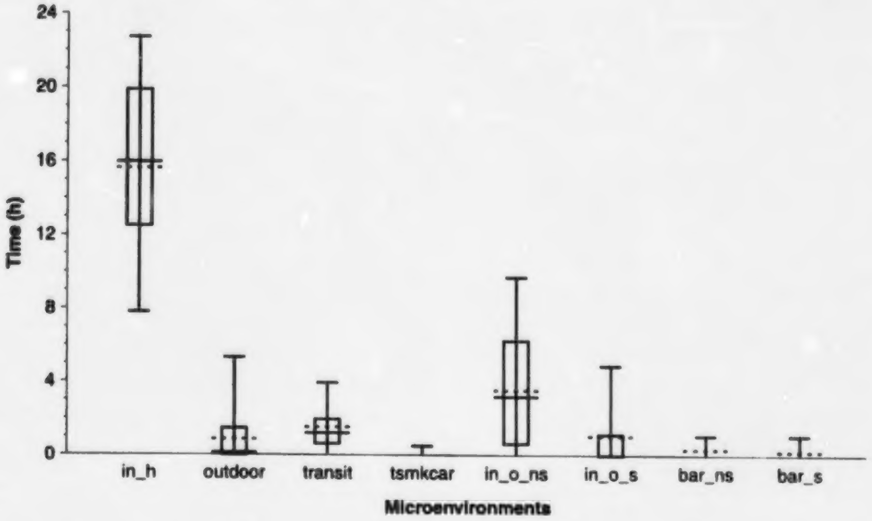
Residential air exchange data in Canada, fit by lognormal distribution, were assumed to be represented by those in the north US for the seasons fall, winter and spring (Özkaynak et al., 1995a). Summer was modelled as being two times greater than in the rest of the year based on summer air exchange rate data for the other region.

6.5.3 Results

The probabilistic PM_{10} exposure simulation model was iteratively run 10,000 times to produce estimated distributions of 24 h average personal, indoor, outdoor and in-transit PM_{10} concentrations. Box plots are used to denote the median, 5th, 25th, 75th and 95th percentiles of the average time spent in various microenvironments. The solid horizontal line is the median, and where appropriate, the dashed line the mean. Figure 6.5 displays the time-activity data (in hours) used in the PM_{10} exposure predictions. Average time spent indoors at home (mean = 15.7 h, nighttime = 10.3 h, daytime = 5.4 h) is the longest time individuals spend within a given microenvironment.

Table 6.16 shows the descriptive statistics for all of the predicted microenvironmental PM_{10} concentrations. Indoor smoking environments dominate PM_{10} concentrations. However, the highest PM_{10} concentrations are estimated in cars with smokers (mean PM_{10} = 71 µg/m^3). Figure 6.6 displays the predicted distributions for 24 h indoor and outdoor microenvironmental concentrations in graphical form. Average residential indoor daytime concentrations are predicted to be about 40 µg/m^3, whereas nighttime levels are lower (25 µg/m^3). In comparison, average outdoor PM_{10} calculated based on ambient measurements (1990-1993), is about 24 µg/m^3.

Figure 6.5 Time-activity Data Used in PM_{10} Exposure Model



in_h	time spent indoors at home
outdoor	time spent outdoors
transit	time in transportation
tsmkcar	time in car spent is presence of a smoker
in_o_ns	time spent indoors other than at home, without smokers
in_o_s	time spent indoors other than at home, with smokers
bar_ns	time spent in smoking free bar/restaurant
bar_s	time spent I bar/restaurant with cigarette smoke

Table 6.16 Summary Statistics of Estimated Microenvironmental PM_{10} Concentrations (μg/m³, 24-h)

	Mean	Std. dev.	Min	5th	25th	50th	75th	95th	Max
Cout	24.4	17.8	0.8	6.0	12.4	19.9	31.1	58.7	148.6
Cin_day	40.4	55.2	0	7.3	14.8	24.8	43.8	124.7	1206.5
Cin_nite	24.8	38.4	0	4.4	8.8	14.3	25.1	76.3	844.8
Cin_o_ns	43.7	39.5	0	0	13.1	34.9	57.2	118.2	483.2
Cin_o_s	52.3	79.8	0	0	0	0	94.1	213.1	744.8
Cbar_ns	7.8	23.2	0	0	0	0	0	54.5	368.6
Cbar_s	29.0	74.0	0	0	0	0	0	204.5	607.2
Ctransit	25.9	21.9	0	0	11.5	21.4	34.9	66.6	175.6
Csmkcar	71.3	157.4	0	0	0	0	99.8	394.0	1333.4

Cout	outdoor concentration
Cin_day	total indoor daytime concentration from all sources
Cin_nite	total indoor nighttime concentration from all sources
Cin_o_ns	concentrations from other indoor no smoking sources
Cin_o_s	concentrations from other indoor smoking sources
Cbar_ns	concentrations in bar/restaurant with no smoking
Cbar_s	concentrations in bar/restaurant with smoking
Ctransit	concentrations in transportation
Csmkcar	concentrations due to smoking in car

Figure 6.6 Predicted PM_{10} Concentrations at Home, Indoors and Outdoors

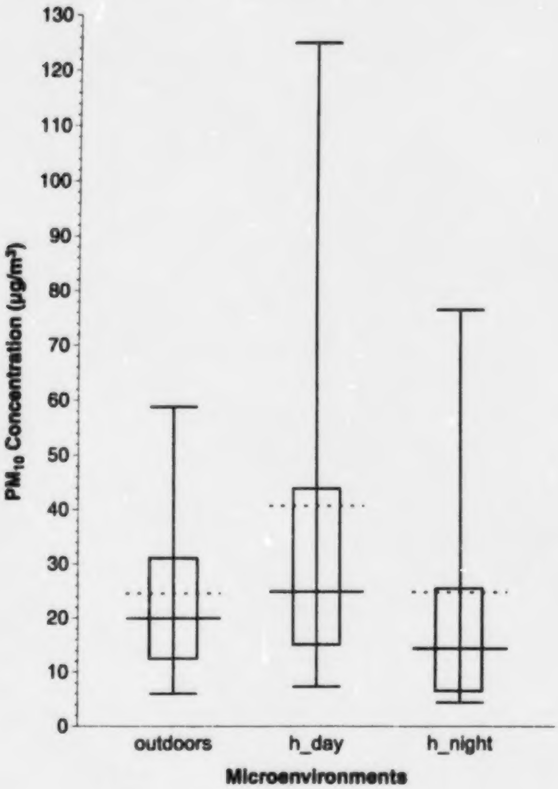
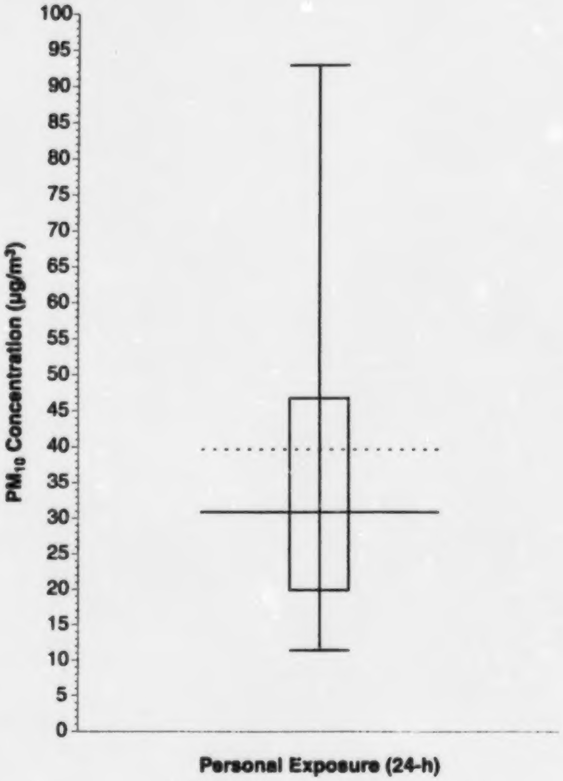


Figure 6.7 National Average Predicted Personal PM_{10} Exposure



Predicted 24 h average personal PM_{10} exposures (across all regions and seasons) is 39 µg/m^3 (Figure 6.7). Median personal PM_{10} exposure is predicted to be 31 µg/m^3 (solid line). The estimated PM_{10} exposure distributions are weighted by the frequency of residences in urban and rural locations, and region-specific population size. Little differences were seen between the predicted exposures in each region. Urban exposure distributions were slightly higher than rural exposures due to differences in the urban versus rural ambient PM_{10} levels. Exposures tended to be slightly higher in the winter than in the spring or summer. A more detailed breakdown of personal exposures by age and gender predicted teenagers and seniors to have lower exposures than the rest of the adult population because of their time-activity profiles and lower smoking rates.

The contributions to personal exposures were calculated; indoors at home and non-residential indoor exposures were predicted to contribute the greatest to personal exposures. Further breakdown of the indoors at home exposures revealed other indoor sources, for example, ETS and unaccounted other sources contribute significantly to residential indoor PM_{10} exposures. Penetration of ambient PM_{10} indoors contributed to indoor levels to a small degree. In-transit or auto-cabin exposures were predicted to be greater than both ambient and in bar/restaurant exposures.

The model predicted vast differences in the personal PM_{10} exposures of individuals exposed to smoke vs non-smoke exposed individuals. The difference is about 20 µg/m^3. Exposure to cooking does not influence the overall distribution of personal PM_{10} exposures.

Uncertainty analysis revealed that uncertainties in the PM_{10} removal or deposition rate, and uncertainties in the estimate of the other indoor residential PM_{10} source strengths strongly influence the uncertainties in the Canadian population distribution of personal PM_{10} exposures. pTEAM data was used to estimate source strengths and infiltration of the high ambient PM10 levels may have biased the statistical model estimates. The uncertainty analysis also revealed that personal PM_{10} exposure variability is strongly correlated (p=0.6) with indoor day or night PM_{10} concentrations.

Additional uncertainties in the estimated population distribution of PM_{10} exposures stem from the use of NHAPS time-activity data rather than Canadian data, which is fairly limited, and the use of non-residential indoor particle concentrations collected in other locations (non-Canadian). Also, the "person cloud" portion of the personal exposure was not incorporated into these estimates. If the pTEAM data, from which the source strengths were estimated, correctly estimated the proportion of "personal cloud" above ambient and indoor particle levels, the predicted microenvironmental PM_{10} exposures would be approximately 10 $\mu g/m^3$ greater.

6.6 DISCUSSION AND CONCLUSIONS

To the extent that microenvironmental concentrations (ambient and non-ambient PM) are different from outdoor concentrations, population exposures (total PM) are different from those estimated by ambient monitoring (ambient PM). Concentrations of particles from different sources are averaged to produce a 24 h personal exposure estimate. The fractions of fine and coarse particles of ambient origin that will be found indoors under equilibrium will tend toward 50% or less, particularly in the winter. The 24 h time-weighted average personal exposure estimate using only ambient data ignores potential exposure to particles in other microenvironments, such as indoors at home, at work, in transit and at other indoor locations. The high correlations between personal exposure and indoor concentrations combined with the time spent indoors indicate that indoor microenvironments are the most important contributors to overall average exposure.

Indoor particles come from the following sources: penetration of outdoor air into the building, indoor sources and generation or resuspension of particles during personal activities such as cooking, dusting and vacuuming (US EPA, 1982a, 1996; Clayton et al., 1993; Wallace et al., 1993; Thatcher and Layton, 1994). The latter source, the personal cloud or "Pigpen effect," explains why personal exposure is greater than indirect estimates using ambient data, or indirect estimates combining indoor and outdoor microenvironments and time-activity information. The increase in particle concentration as a result of a person occupying the microenvironment is overlooked. Time-weighted average exposure estimates often do not capture the increase in particles due to resuspension, while in transit, and as a result of smoking or exposure to ETS.

The additional particle sources vary independently of the centrally located fixed ambient monitor (FAM). Limited data on particle concentrations with short-term averaging times reveal that peak concentrations occur indoors, outdoors, in traffic and while smoking. These sources are not represented by current ambient monitoring networks. Therefore, the relationship between ambient data and indoor and personal exposure is not linearly related. If there is a lot of variability in particle concentrations from sources poorly correlated with FAMs, then the percentage of variance in personal exposure to total PM that can be explained by the FAM data is small.

Epidemiological studies provide concentration-response information rather than exposure- or dose-response data by not linking the source of particulate matter with the exposure process and adverse outcome. Effects measured at a given concentration do not constitute a full risk assessment, because the number of people exposed, and the concentration they are exposed to is not known. The result is to underestimate the associated risks and their statistical significance. However, insufficient data exist to determine whether short-term exposure to high levels of particles results in the same degree of health impact as do longer-term, lower-concentration exposures.

Epidemiology studies are based on PM measurements at a central site (FAM). PM concentrations at FAM sites can represent the exposure of the people in the community to PM of ambient origin. However, comparing parts of the distribution of exposure predicted using urban NAPS sites with the predicted exposures from the probabilistic exposure model clearly shows ambient data underestimate average population exposures to total PM (Table 6.17). Although estimates of exposure to $PM_{2.5}$ have not been predicted through exposure modelling, using the mean ratio of ambient $PM_{2.5}/PM_{10}$ presented in Table 5.20 (0.50), population exposure estimates to fine particles were estimated.

Table 6.17 Predicted Mean, Median, High End Exposure Estimates to Ambient PM ($\mu g/m^3$)

	mean	SD	median	P95	max
Urban Ambient PM_{10} Data	27.6	16.3	24.0	58.0	175.0
Predicted PM_{10} Exposures	39.4	33.5	30.9	93.0	682.0
Urban Ambient $PM_{2.5}$ Data	13.9	9.5	11.0	32.2	89.0
Calculated $PM_{2.5}$ Exposures	19.7	16.75	15.5	46.5	341

Given the current information, concluding that the ambient data alone can represent the lower range in the distribution of total particle exposures is reasonable.

6.7 FUTURE REQUIREMENTS

Sources of PM_{10} and $PM_{2.5}$ vary among different provinces as well as between various indoor and outdoor environments. Consequently, the contributions of ambient particles to indoor particle concentrations and subsequently, to total personal exposures will vary by different geographic and climate zones.

It is recommended that special studies be carried out to determine the exact nature of the indoor sources of indoor air particles.

It is recommended that special studies be carried out to determine the penetration rate of ambient particles indoors, specifically in colder climates, where the air exchange rates are a function of ambient temperature.

7 THE AESTHETIC EFFECTS OF PARTICULATE MATTER (VISIBILITY)

Increasing concentrations of fine particles and gases in the atmosphere often result in reduced visual range (or degradation of visibility), by creating a haze that obscures the clarity and structure of what is observed. Visibility is one of the most readily perceived indicators by the public of poor air quality, and is often cited as a concern in urban and wilderness areas. Historically, visual range has been an important weather parameter relevant to the safe operation of aircraft. Visual ranges less than 24.1 km ($PM_{2.5} \approx 18\ \mu g/m^3$) present a hazard to aircraft using airports; visual ranges of less than 1 km may cause airports to close.

Prevention of visibility deterioration in cities, national parks and wilderness areas is also relevant due to potential loss of tourism and quality of life. Although the aesthetic effects of visibility degradation are subjective in nature, several perception studies have attempted to quantify the significance of reduced visual range. These include determining what is considered "acceptable" visual range (Pryor et al., 1994b), assessing the economic value of incremental changes in visual range (Chestnut and Latimer, 1994), and estimating the amount of time visitors are willing to drive the see certain vistas (Malm et al., 1984).

This chapter will focus on the relationship of TSP, PM_{10}, and $PM_{2.5}$ to visual range. More detailed reviews on effects of particles on visual range are available: Malm, 1979; Pueschel, 1983; Hoff, 1983; Hoff and Barrie, 1985; Sloane, 1986; White, 1986; Trijonis et al., 1990; Laulainen, 1993.

7.1 THE RELATIONSHIP BETWEEN PARTICULATE MATTER AND VISUAL RANGE

There is a direct relationship between the amount of particulate matter in the atmosphere and the ability of the human eye to see through the atmosphere. Perception of distant objects involves contrast, where the contrast of the distant object against the background (usually the sky) decreases as the distance between the object and the observer increases. Visual range is the measure of transparency of the atmosphere, defined as the distance at which the contrast of the target is equal to the threshold contrast value for the human eye.

Physically, visual range is a function of the light scattering and absorption properties of gas molecules and particles. The light extinction coefficient (b_{ext}) is a measure of light attenuation as a result of scattering and absorption by gas molecules and aerosol particles along the direction of propagation of the light beam. The Beer-Lambert law mathematically expresses this relationship:

$$\begin{aligned} d_{II} &= -b_{ext}dx \\ &= -(b_{scat} + b_{abs})dx \\ &= -(b_{RG} + b_{sp} + b_{ag} + b_{ap})dx \end{aligned} \tag{7.1}$$

$$b_{scat} = b_{RG} + b_{sp} \tag{7.2}$$

$$b_{abs} = b_{ag} + b_{ap} \tag{7.3}$$

where:

b_{ext} = light extinction coefficient (distance^{-1}),

b_{scat} = light scattering coefficient (distance^{-1}),

b_{abs} = light absorption coefficient (distance^{-1}),

I = intensity of the light beam,

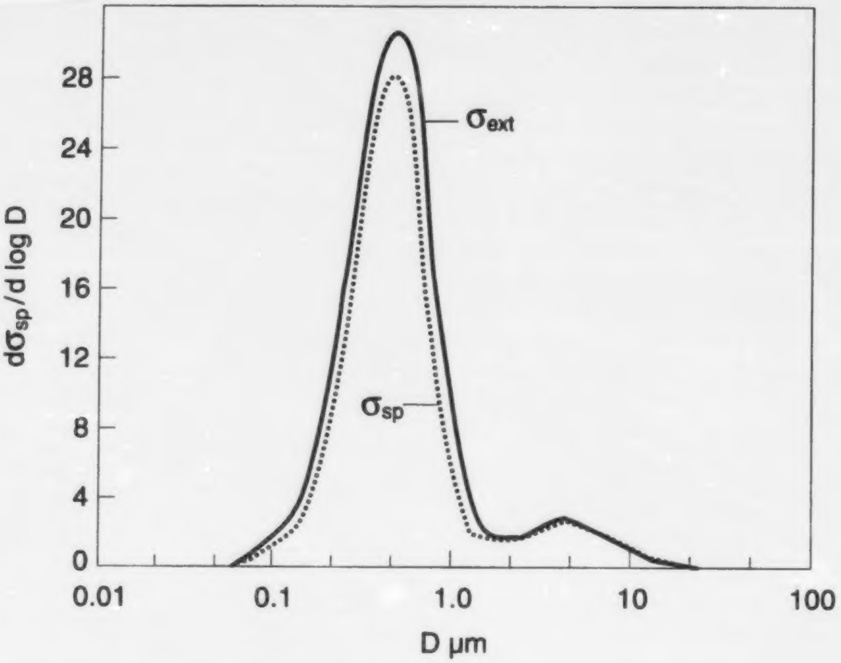
dI = the change of intensity as the light beam traverses a distance

$b_{RG} + b_{sp}$ = the air molecules (Rayleigh) and particle light scattering coefficients in units of distance^{-1}, and

$b_{ag} + b_{ap}$ = the gas molecule and particle light absorption coefficients in units of distance^{-1}.

Equation 7.1 shows that the light extinction coefficient, b_{ext}, is the sum of the light scattering and light absorption coefficients (b_{scat} and b_{abs}, respectively). Waggoner et al. (1981) showed that particle size is the most important factor in determining the relationship between particle mass and b_{scat}. Figure 7.1 shows the particle extinction cross sections for light

Figure 7.1 Particle Extinction Cross Sections for Extinction (σ_{ext}) and Scattering (σ_{sp}) as a Function of Particle Size Diameter (from Waggoner et al., 1981)



extinction and scattering as a function of particle size diameter. This figure also shows that light scattering is the dominant component of light extinction (i.e., light absorption is usually relatively small). Waggoner and Weiss (1980) showed that single particle optical cross section per unit particle volume reaches a maximum for particles with diameters in the range of 0.3-0.7 μm. Thus, fine particles (<2.5 μm) dominate light scattering and hence light extinction. In other words, fine particles ($PM_{2.5}$) and submicrometre particles ($PM_{<1}$) in particular are most effective in reducing visual range.

Visual range (*VR*) is determined as follows:

$$VR = \ln(C(0) / Cd) / b_{ext} \qquad (7.4)$$

where:

b_{ext} = the light extinction coefficient in units of distance^{-1},

$C(0)$ = the object's inherent contrast (contrast at zero distance), and

Cd = the threshold for detection of the object's contrast.

If the object is black then $C(0) = -1$ and the threshold for detection of the object's contrast has been estimated to be 2% ($|Cd|=0.02$). Equation 7.4 can be simplified for a perfect (i.e., non reflecting) black object to the Koschmieder equation:

$$VR = 3.91 / b_{ext} \qquad (7.5)$$

where visual range is in distance units and the factor 3.91 is based upon a 2% threshold value for distinction of an object from background (Charlson et al., 1968). Equation 7.5, the Koschmieder equation, describes the relationship between a measured atmospheric parameter, b_{ext}, and human perception of visual range. The Koschmieder equation incorporates several assumptions: the atmosphere is homogeneous such that scattering and absorption of radiation are the same everywhere, sky brightness is the same at the object, the background and the observer (cloudless sky), and the viewing distance is horizontal, i.e., the earth's curvature is ignored (Seinfeld, 1986). A number of authors have used this relationship to compare measured light-scattering coefficients with visual range studies (Dzubay et al., 1982; Pitchford and Malm, 1994; Pryor, 1996).

7.2 PARTICLE CHARACTERISTICS AND THE LIGHT EXTINCTION COEFFICIENT

Particle mass, hygroscopicity and particle composition all affect the degree to which ambient particulate matter may affect visual range. Light scattering is the sum of Rayleigh scattering, b_{RG}, (due to gaseous molecules) and particle scattering, b_{sp}. In a virtually particle free atmosphere Rayleigh scattering predominates. Rayleigh scattering at sea level is $1.0 \times 10^{-5} m^{-1}$ at 550 nm and decreases from $0.88 \times 10^{-5} m^{-1}$ to $0.61 \times 10^{-5} m^{-1}$ for altitudes of 1000 to 4000 m (Waggoner et al., 1981). As particle concentration increases, particle scattering will increase until $b_{sp} \geq b_{RG}$.

Light scattering dominates light absorption except where there are light absorbing particles or gases present. Graphitic or elemental carbon (commonly known as soot) is very efficient at absorbing light. Particle absorption (b_{ap}) is $\leq$10% of particle scattering (b_{sp}) in rural areas but can be nearly equal to particle scattering in urban areas where graphite carbon is present (Waggoner and Weiss, 1980). NO_2 is the only light absorbing gaseous species present that is optically significant. This gas absorbs light wavelengths at the blue end of the visible spectrum, resulting in a brownish colour. The presence of NO_2 is important in studies on plume optics but not in the case of a well-mixed layer (Waggoner, 1980).

Measurements of visual range taken with a nephelometer are expressed in terms of the scattering coefficient (b_{scat}), while measurements done using transmissometers are expressed in terms of b_{ext}. In order to calculate visual range (equations 7.4 & 7.5) the value of b_{ext} must be measured or calculated from the ratio of b_{scat}/b_{ext}, which is assumed to be constant. As the presence of graphite carbon influences the particle absorption coefficient, b_{abs}, and thus b_{ext} in urban areas, the ratio of b_{scat}/b_{ext} is estimated separately for urban and rural areas. Waggoner et al. (1981) calculated a ratio of b_{scat}/b_{ext} for two locations near Buffalo National River in northeastern Arkansas from b_{scat} measured with a nephelometer and b_{ext} calculated from equation 7.5 with telephotometer measurements of sky and target radiance. The correlation of b_{scat} and b_{ext} from these instruments ranged from 0.84 to 0.99. There was a higher correlation for cloud-free days. A ratio of b_{scat}/b_{ext} of was calculated for all days (0.77 to 1.20), for cloud-free days (0.8 to 1.04), and was dependent on time of the day. On average for this rural site, b_{scat} was about 0.9 of b_{ext}. For sites located in the metropolitan area of Denver, the ratio of mean b_{scat}/b_{ext} was 0.7 (Middleton et al., 1984), confirming the larger role of absorption in light extinction in urban areas. Similarly, in Houston, Texas, the ratio of b_{scat}/b_{ext} was 0.77 (Dzubay et al., 1982), and in the Fraser Valley, the mean ratio of b_{scat}/b_{ext} was 0.6 (Chilliwack site; Pryor et al., 1995a). From these values, a reasonable estimate of b_{ext} can be determined from:

$$b_{scat}/b_{ext} = 0.7 \text{ in urban areas, and} \qquad (7.6)$$

$$b_{scat}/b_{ext} = 0.9 \text{ in rural areas.} \qquad (7.7)$$

7.2.1 Relationship Between Particle Mass and Light Extinction and Scattering Coefficients

A number of studies have found that there is a linear relationship between particle mass and b_{ext} or b_{scat}. Table 7.1 shows a range in values determined for the ratio of b_{ext} or b_{scat} to particle mass. In early studies, Charlson et al. (1968) suggested that TSP/b_{scat} ranges from 0.2 to 0.7 g/m^2 (b_{scat}/TSP = 1.4-5 m^2/g). For particles of radius < 50 μm, the ratio of b_{scat}/TSP was calculated to be 1.6-2 m^2/g (Horvath and Charlson, 1969). Recent studies have focused on fine particles, of size range 0.1-2.5 μm, due to their efficiency at light extinction. Consequently, a stronger relationship was found when fine particulate mass is used instead of TSP (Table 7.1). Waggoner et al. (1981) compared Charlson et al. (1968) relationship of TSP and b_{scat} at a number of US urban sites with that of fine particulate mass <2.5 μm. A linear relation was observed between TSP and b_{scat}, with a ratio of b_{scat}/TSP ≈ 2, but there was more variation in the ratio than for b_{scat}/fine particulate. Waggoner et al. (1981) found that fine particulate mass and b_{scat} has a correlation >0.95. The relationship of b_{scat}/fine mass has been shown to be 3.1±0.2 m^2/g for a number of urban and rural sites in the United States (Waggoner and Weiss, 1980).

The ratios of b_{ext}/mass and b_{scat}/mass generally decrease when mass includes coarse particles (PM_{10} or TSP). A stronger correlation has been shown to exist between fine particulate mass and light scattering, therefore, linear relationships derived for coarse particles and light scattering can be expected to have more error associated with them. For some US sites (Lewis et al., 1986; White and Macias, 1990), ratios of b_{ext}/fine particle mass in the range of 3.3-4.3 m^2/g have been observed. These

Table 7.1 Comparison of Relationship of Coefficient of Scattering or Extinction with Particle Mass

Site	Ratio	Value (m^2/g)	Reference
Mesa Verde, CO (pristine)	$b_{scat}/PM_{2.5}$	2.94	Waggoner and Weiss (1980), Waggoner et al. (1981)
Seattle, WA (residential)		3.13	
Seattle, WA (industrial)		3.23	
Puget Island, WA (rural)		3.03	
Portland, OR (industrial)		3.23	
Denver, CO	$b_{ext}/PM_{2.5}$	4.3	Lewis et al. (1986)
	b_{ext}/PM_{15}	2.1	
Spirit Mtn., NV (rural US)	$b_{ext}/PM_{2.5}$	3.3	White and Macias (1990)
	b_{ext}/PM_{15}	1.56	
Urban US sites	b_{scat}/TSP	1.4-5	Charlson et al. (1968)
from regression line	b_{scat}/TSP	2.6	
Airport urban data and TSP	b_{ext}/TSP	2	Noll et al. (1968)
Urban	$b_{p,scat}/TSP$	3.4	Kretzschmar, (1975)
Calculated for particles $r < 50$ µm	$b_{scat}/mass$	1.6-2.0	Horvath and Charlson (1969)
Santiago, Chile	b_{ext}/PM_{10}	5	Trier and Horvath (1993)
Berlin, Germany	b_{ext}/PM_{10}	6.0	Heits and Israel (1982)
Vancouver, B.C.	$b_{scat}/PM_{2.5}$	3.0	Guise-Bagley and Hoff (1993)
Houston, TX	$b_{scat}/PM_{2.5}$	3.52	Dzubay et al. (1982)

ratios are only slightly higher than the ratio of b_{scat}/fine particle mass of 3.1 m^2/g reported by Waggoner and Weiss (1980). A value of 3.1 m^2/g was used for b_{scat}/fine mass in this report as it represents an average of a large number of urban and rural sites and is similar to measurements taken at Vancouver (3.0 m^2/g). Currently, site specific data for b_{scat}/fine particulate mass is not available for Canadian visibility monitoring sites (Waterton, Egbert, St. Andrews), thus for the purposes of this assessment:

$$b_{scat}/PM_{2.5} = 3.1\ m^2/g \qquad (7.8)$$

Pitchford (1982) showed that coarse particles could also disrupt visibility. Coarse particles were shown to be one-half to one-third as effective as fine particles in disrupting visibility. However, in some areas, where coarse particle concentrations are unusually high, they can contribute from 30 to 80% of total light extinction (Pitchford, 1982). Following Malm (1993), if:

$b_{scat} / PM_{2.5} = 3.1\ m^2/g$ and $b_{scat} / (PM_{10} - PM_{2.5}) = 0.6$ then

$PM_{2.5} = 0.6\ PM_{10} - 0.6\ PM_{2.5} = b_{scat}$.

Thus, for the contributions to reduced visual range to be equal, PM_{10} must equal $6 \times PM_{2.5}$. More simply put:

$$b_{scat} = 3.1\ PM_{2.5} + 0.6\ (PM_{10} - PM_{2.5}) \qquad (7.9)$$

Site specific data is preferable for deriving relationships between light extinction or scattering and particle mass given the variability among sites in particle characteristics and particle mass.

7.2.2 Effects of Particle Composition on Visual Range

Sulphate, nitrate, soil, organic carbon and elemental carbon dominate fine particulate matter composition (Pryor, 1994; Malm, 1992). A number of studies have examined the relative importance of chemical species to reduced visual range (Hidy and Brock, 1971; Cass, 1976; White and Roberts, 1977; Leader et al., 1979; Trijonis, 1980, 1982; Groblicki et al., 1981; Dzubay et al., 1982; Malm, 1992; Sisler and Malm, 1994). These studies found that sulphates are the largest contributor to visual range reduction, and that nitrates are also significant contributors but less efficient per unit mass than sulphates in light scattering.

Appel et al. (1985) showed however, that in some areas, nitrate contributions to light scattering were greater than those of sulphates (sulphate 30.4% and nitrate 45.1%; San Jose, Riverside and downtown Los Angeles). Pryor et al. (1996a) found that for the Fraser Valley, fine mass was dominated by a secondary organic component, but sulphate and ammonium nitrate dominated light scattering. Particle-phase organic compounds are relatively inefficient contributors to light scattering. Elemental or black carbon contributes to light absorption and, to a small extent, light scattering. Measurements from the IMPROVE network in the United States show that ammonium sulphate and organic carbon are the largest contributors to fine particulate mass and reduced visibility (Malm, 1992).

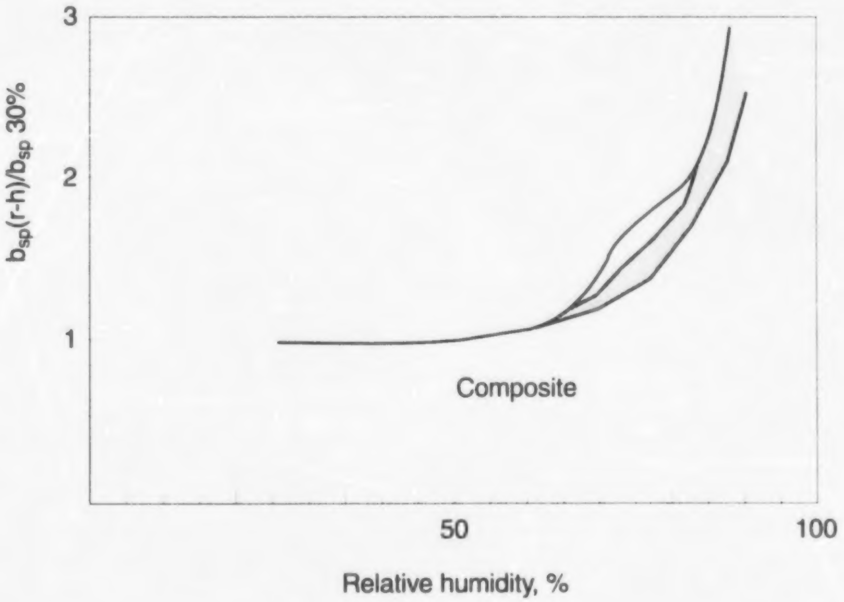
7.2.3 Effects of Relative Humidity on Light Extinction and Scattering Coefficients

Relative humidity influences particle light extinction. When relative humidity exceeds 70%, light scattering efficiencies begin to increase (Charlson et al., 1984). As humidity increases to 95% or more, water soluble components of the fine aerosol can absorb water and swell to more than seven times their dry radius. Covert et al. (1980) showed that scattering increases by a factor of two to three times at many US sites for a change in relative humidity of 30 to 80%, as shown in Figure 7.2. Pitchford and McMurry (1994) studies further confirm this behaviour. The effect of humidity on light scattering properties is also very dependent on chemical and microphysical variables. Components of fine particles, such as sulphates (ammonium sulphates), nitrates (ammonium nitrate), organics, elemental carbon and soil dust, will vary in their ability to absorb water. The hygroscopic fraction of the aerosol will affect visual range largely in regions of Canada with higher relative humidity.

7.3 PARTICULATE MATTER LEVELS AND ESTIMATED VISUAL RANGES

An estimate of visual range and particle levels can be determined using the Koschmieder equation (equation 7.5) by assuming a ratio of light scattering coefficient to fine particle mass of 3.1 m^2/g and ratios of b_{ext} to b_{scat} given in equations 7.6 and 7.7 for urban and rural sites. Brook et al. (1996) calculated that $PM_{2.5}$ accounted for 49% of PM_{10} and PM_{10} accounted for 44% of the TSP, on average,

Figure 7.2 Measured Enhancement of Scattering (B_{sp}) as a Function of Relative Humidity.



Note: Hatched area contains the complete range of ambient measurements illustrating the difference between sites in the US West and Midwest (Covert et al., 1980).

across Canadian sites. (Note: the majority of these sites are urban, therefore the following relationships may better characterize urban than rural locations.) Based on the linear regression of $PM_{2.5}$ and PM_{10} with TSP for all Canadian sites, the equivalent PM_{10} and $PM_{2.5}$ levels can be calculated as follows:

equivalent $PM_{10} = 0.44 \times TSP$ (7.10)

equivalent $PM_{2.5}=0.21 \times TSP$ (7.11)

equivalent $PM_{2.5}=0.73 + 0.49 \times PM_{10}$ (7.12)

where particle concentrations are in units of μg/m^3. Visual range and b_{scat} were calculated from the fine particle masses (units g/m^3) and are presented in Table 7.2. b_{scat}, and hence b_{ext}, are estimates, and the visual range calculation assumes that the particles are non-absorbing, a 2% threshold value for distinction of an object from the background and relative humidity <70%. Actual visual ranges may be significantly less in regions with relative humidity in excess of 70%.

7.3.1 Visual Range Estimates for Current PM Air Quality Criteria

Table 7.2 lists the current Canadian TSP ambient air quality objectives, equivalent PM_{10} and $PM_{2.5}$ levels (Dann, 1994), 24 h and annual US PM_{10} and $PM_{2.5}$ standards and the 24 h British Columbia objectives and Ontario (interim) PM_{10} standard. The PM air quality criteria in Table 7.2 were converted to PM_{10} and $PM_{2.5}$ equivalents using the relationships presented above. Visual range and b_{scat} was calculated using the fine particle mass equivalents. As previously stated, the size fraction of particles and components primarily associated with light scattering causing reduced visibility is <2.5 μm (dominated by particles with diameter 0.3-0.7 μm).

7.3.2 Visual Range Estimates for Estimated Natural Visibility

Table 7.3 shows estimated ranges of natural visibility (and b_{scat}) at a number of non-urban sites in the United States and Canada (Trijonis, 1982; Malm, 1992; Hoff et al., 1997; Fox et al., 1997) and calculated fine particulate levels. b_{scat} is determined from nephelometer readings and is used to calculate visual range based upon equations 7.7 and 7.5. The fine particulate levels are estimated from equation 7.8 for a number of urban and rural sites in the United States (Waggoner and Weiss, 1980).

Natural visual ranges in Canada, estimated from the 10th percentile in the b_{scat} distribution, are 86 to 350 km and from these, estimated natural $PM_{2.5}$ levels

Table 7.2 Visibility Equivalents of Current Canadian, US and California Particulate Matter Objectives and Guidelines

Particulate standard or objective (μg/m^3)	Equivalent PM_{10} (μg/m^3)	Equivalent $PM_{2.5}$ (μg/m^3)	Visual Range (km)		b_{scat} ($\times 10^6$/m)
			Rural	Urban	
TSP 400 24 h – Cdn TSP Tolerable level	180	84	14	11	260
TSP 120 24 h – Cdn Acceptable 24 h Level	53	25	45	35	78
TSP 70 annual – Cdn Acceptable Annual Level	31	15	77	60	46
PM_{10} 150 24 h – US NAAQS	150	74	15	12	230
PM_{10} 50 24 h – BC + Ontario (interim) objective and US annual NAAQS	50	25	45	35	78
$PM_{2.5}$ 65 24 h – New US NAAQS		65	14	18	201
$PM_{2.5}$ 15 annual – New US NAAQS		15	76	59	47
Lower range mean Cdn urban and rural $PM_{2.5}$ levels (Dann, 1994)		8	140	110	25
Upper range mean Cdn rural $PM_{2.5}$ levels (Dann, 1994)		11	100	80	34
Upper range mean Cdn urban $PM_{2.5}$ levels (Dann, 1994)		22	50	40	68

are 3-13 µg/m^3 (Table 7.3). Natural visual range exists in regions not directly impacted by anthropogenic emissions. Western Canada and eastern Canada have estimated natural levels of $PM_{2.5} \leq 6$ µg/m^3, while southeastern Canada has a higher natural $PM_{2.5}$ level of 10-13 µg/m^3. A 10% change in visual range is considered the minimum observable incremental change (see section 7.4). Given that there is a linear relationship between visual range and $PM_{2.5}$, a 10% increase in $PM_{2.5}$ levels above background levels (Table 7.3) represents the change required to detect effects (i.e., decreased visibility). Thus, in western and eastern Canada, impacts on visual range, above background levels, would be detectable when $PM_{2.5}$ levels exceed 6 to 7 µg/m^3 and for southeastern Canada, 14 µg/m^3, using upper limits of estimated $PM_{2.5}$ levels.

7.3.3 Visual Range Estimates for Canadian Sites

As detailed in Chapter 5, the relative distribution of PM_{10} to TSP ratios and $PM_{2.5}$ to TSP ratios is a function of geographic location as well as proximity of the monitoring station to local sources. At Canadian sites, the mean ratio of PM_{10} to TSP ranges from 0.36 to 0.79. PM_{10} to TSP ratios are significantly lower at Prairie sites than at eastern Canadian and Vancouver sites (Prairie sites, PM_{10}/TSP = 0.36-0.53, compared with Vancouver and Halifax, PM_{10}/TSP = 0.67-0.77). Mean $PM_{2.5}$ to TSP ratios range from 0.20 to 0.50. The majority of NAPS sites show 50 to 60% of PM_{10} as $PM_{2.5}$ size fraction (Table 7.4). Prairie urban sites such as Edmonton, Winnipeg, and Calgary had less than 50% of PM_{10} as $PM_{2.5}$ (Dann, 1994). The values for PM_{10}/TSP and $PM_{2.5}$/TSP ratios presented here differ slightly from those in chapter 5, Table 5.20 as somewhat different databases were used in each case. At urban sites across Canada, estimated visual range extends from 34 to 73 km, and from 93 to 136 km at rural sites.

As $PM_{2.5}$ is primarily responsible for the scattering of light, visual ranges at current TSP air quality objective levels were estimated for specific urban sites using a $PM_{2.5}$ level estimated from the $PM_{2.5}$/TSP ratio for each site (Table 7.5). Equivalent PM_{10} levels were also calculated for the NAPs sites based on ratios of PM_{10}/TSP for each of the sites. The Prairie sites were selected as they had the lowest values of PM_{10}/TSP and $PM_{2.5}$/TSP, while Vancouver and Halifax had the highest values. This table shows that visual range in the Prairies (which had the lowest values of PM_{10}/TSP and $PM_{2.5}$/TSP) are much greater than in Vancouver or Halifax (which had the highest values of PM_{10}/TSP and $PM_{2.5}$/TSP), for a specified TSP level. For example, for PM_{10} levels ranging from 22-32 µg/m^3 (equivalent to TSP levels of 60 µg/m^3), and assuming $b_{scat}/b_{ext} = 0.7$ for urban sites (equation 7.6), visual ranges of 64 to 92 km versus 33 to 36 km occur at Prairie versus Vancouver and Halifax sites respectively.

Table 7.3 Estimated Natural Visual Ranges in North America

Site location (time period)		Visual range, km (estimated from b_{scat} (x 10^6/m))	Estimated $PM_{2.5}$ (µg/m^3)	Reference
Natural, eastern US		100-130	8.8-12.0	Malm (1992)
Natural, western US		170-180	6.4-6.7	Malm (1992)
Visibility observations, western Great Basin & Desert southwest US		100-130	8.8-12.0	Trijonis (1982)
Western US (Glacier National Park)	Seasonal best 10% of b_{scat} 93-94	150-160(22-23)	7.0-7.5	Fox et al. (1997)
Western Canada (Waterton, Alberta)	Seasonal best 10% of b_{scat} 93-95	210-350(10-17)	3.2-5.5	Hoff et al. (1997) Fox et al. (1997)
Southeastern Canada (Egbert, Ontario)	Seasonal best 10% of b_{scat} 93-95	86-120(30-41)	9.7-13.0	Hoff et al. (1997)
Eastern Canada (St. Andrews, N.B.)	Seasonal best 10% of b_{scat} 95	185-210(17-19)	5.5-6.1	Hoff et al. (1997)

Note: VR calculated from $b_{scat}/b_{ext} = 0.9$, $VR = 3.91/b_{ext}$, and $PM_{2.5}$ calculated from $b_{scat}/PM_{2.5} = 3.1$m^2/g.

Table 7.4 Estimated Visual Ranges Associated with Mean 24 h $PM_{2.5}$ and PM_{10} Levels (1984-1993)

Station no.	City	Mean $PM_{2.5}$ (µg/m³)	Mean PM_{10} (µg/m³)	Estimated Visual Range (km)
40203	Saint John	10.7	18.3	73
30101	Halifax	14.3	26.0	53
30501	Kejimkujik	7.5	11.8	136
50104	Montréal	16.1	28.1	48
50109	Montréal	21.4	45.7	34
50307	Québec City	12.1	24.3	61
54101	Sutton	7.9	11.6	132
60104	Ottawa	12.7	22.8	60
60204	Windsor	18.6	32.7	41
60211	Windsor	17.2	30.4	45
604117	Toronto	16.7	27.9	47
60424	Toronto	17.6	29.3	44
61901	Walpole Is.	18.1	30.3	43
64401	Egbert	10.9	17.9	93
70119	Winnipeg	10.4	29.0	63
90130	Edmonton	10.5	27.3	64
90204	Calgary	14.4	32.1	50
90227	Calgary	10.1	24.8	68
100106	Vancouver	17.3	27.7	46
100111	Vancouver	15.8	27.2	49
100118	Vancouver	14.1	23.0	56
100303	Victoria	11.6	17.9	69

1. Visibility calculated from equivalent $PM_{2.5}$ levels,$b_{scat}/PM_{2.5}$ = 3.1m²/g, b_{scat}/b_{ext} =0.7, and VR = 3.91/b_{ext} .
2. b_{scat}/b_{ext} = 0.9 was used for the three rural sites of Kejimkujik, Sutton and Egbert.
3. These estimates include contributions to reduced visual range from both the fine and coarse fraction according to equation 7.9.

Table 7.5 Visual Range Estimates for PM_{10} and $PM_{2.5}$ Estimated Levels (µg/m³) Based on TSP NAAQO

TSP (µg/m³)	Prairie Sites			Vancouver and Halifax		
	Equivalent PM_{10} (µg/m³)	Equivalent $PM_{2.5}$ (µg/m³)	Visibility (km) estimate from $PM_{2.5}$	Equivalent PM_{10} (µg/m³)	Equivalent $PM_{2.5}$ (µg/m³)	Visibility (km) estimated from $PM_{2.5}$
120	43-64	19-28	32-46	80-92	49-53	17-18
70	25-37	11-16	55-79	47-54	29-31	29-31
60	22-32	10-14	64-92	40-46	25-26	33-36

1. Canadian TSP air quality objectives: annual mean 70 µg/m³ (geometric mean 60 µg/m³), 24 h 120 µg/m³.
2. $PM_{2.5}$ calculated from $PM_{2.5}$/TSP of 0.16-0.23 for Prairies and 0.41-0.44 for Vancouver and Halifax.
3. Visibility calculated from equivalent $PM_{2.5}$ levels,$b_{scat}/PM_{2.5}$ = 3.1m²/g, b_{scat}/b_{ext} =0.7, and VR = 3.91/b_{ext} .

7.4 PUBLIC PERCEPTION OF DETERIORATION IN VISIBILITY

In addition to the regional differences in visual range for a given particulate loading, there are regional differences in the public's response to changes in visual air quality. Differences in public perception of unacceptable visibility may be related to the aesthetic appeal of environments and vistas viewed, as the public may be more willing to pay for protection of a wilderness area or unique urban skyline. Perception of colour, contrast of markers with background (usually sky) and perception of contrast detail in the atmosphere will affect decisions of acceptable or unacceptable visibility. Since acceptable visibility is subjective, it is important to collect feedback from a population sample that would represent the entire spectrum of "the public". Public perception studies typically use photographs of a limited number of scenes taken during similar times of the day to determine what is an acceptable visual range or what change in visual range is acceptable. Public perception studies have been conducted in wilderness areas and national parks in the United States (Latimer et al., 1981) and the Denver region (Ely et al., 1991; Middleton and Burns, 1991). Public perception studies take different approaches (Malm et al., 1981; Middleton et al., 1985; Ely et al., 1991). Some place a monetary value upon an incremental improvement in visibility (Chestnut and Latimer, 1994). Other studies have examined the relationship between acceptable visual range thresholds and population response of what is acceptable or unacceptable visibility (Pryor et al., 1996a).

The subjective nature of perceived visual range and the need to determine the tolerance of society to changes in visual range (which can vary significantly in different regions) prompted Pitchford and Malm (1994) to propose a scale based on perceived changes in visibility. It's referred to as the deciview scale, *dv*, where:

$$
\begin{aligned}
dv &= 10 \times \ln(b_{ext}/0.01\ \mathrm{km}^{-1}) \\
&= 10 \times \ln(391/VR) \qquad (7.13)
\end{aligned}
$$

A change in 1.0 *dv* corresponds to a 10% change in b_{ext}, which is a small but noticeable scenic change under many circumstances:

$$
\begin{aligned}
dv &= 10 \ln (b_{ext}/0.01) \text{ and } d(dv)/db_{ext} \\
&= 10\,(0.01/b_{ext})(1/0.01) \\
&= 10/b_{ext}
\end{aligned}
$$

therefore $d(dv) = 10\ db/b_{ext}$ or if $d(dv) = \pm 1$ then $db/b_{ext} = \pm 0.1$ or 10% change in b_{ext}.

Figure 7.3 illustrates the relationship of haziness and extinction coefficient or standard visual range (Pitchford and Malm, 1994).

Figure 7.3 Visual Range and Extinction Coefficient as a Function of Haziness Expressed in Deciview (from Pitchford and Malm, 1994)

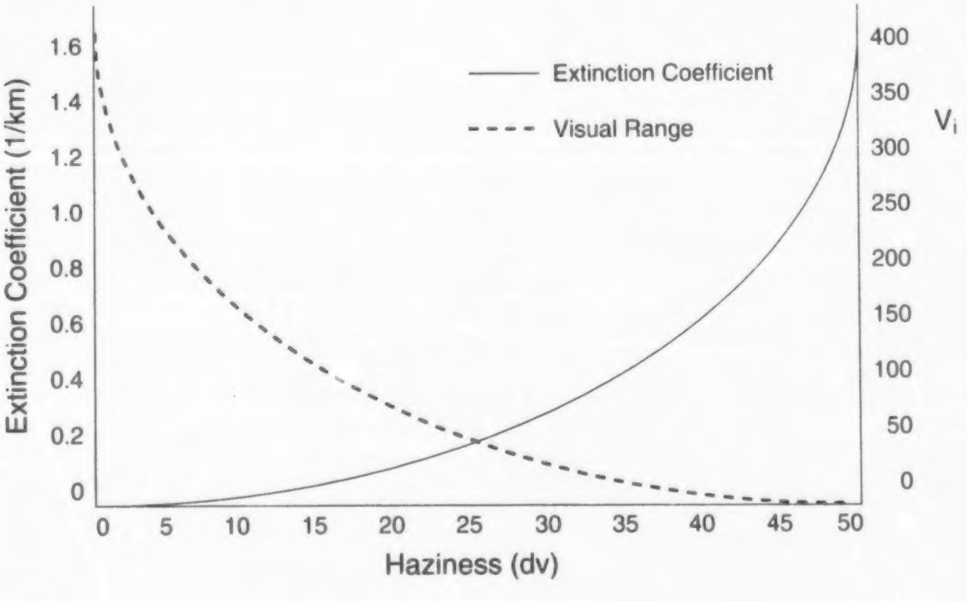
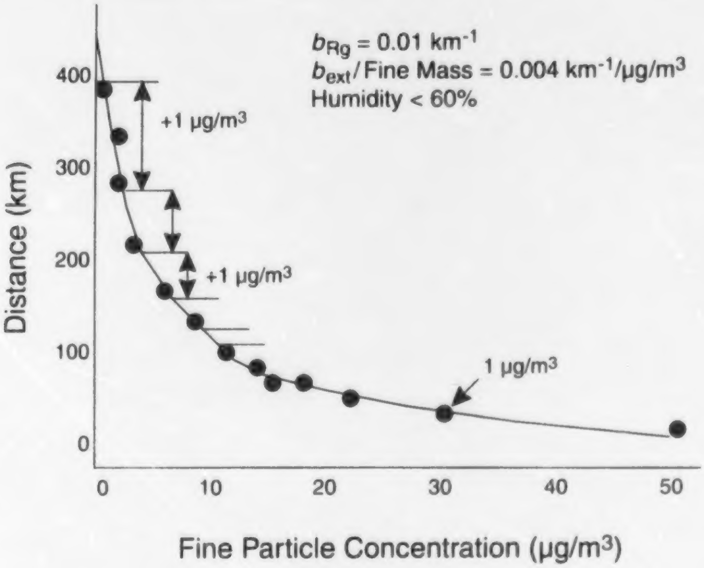


Figure 7.4 Effects of Fine Particle Increments on Calculated Visual Range (from Littlejohn et al., 1981)



A 10% change from existing fine particle levels results in a noticeable change in visibility, illustrated in Figure 7.4. Again, the figure demonstrates that when fine particle levels are low, an addition of 1 µg/m^3 will result in a larger change in visual range than at higher particle levels (Littlejohn et al., 1981).

Figure 7.4 shows that at a level of 10 µg/m^3, a 1 µg/m^3 change would be noticeable. At a level of 30 µg/m^3, a 3 µg/m^3 change would be required to cause a noticeable change in visibility. Consequently, in regions of Canada such as the Prairies, where fine particle levels are lower, a noticeable change in visibility will occur at a lower particle level than for Vancouver or Halifax. Figure 7.4 also shows that the addition of 1 µg/m^3 to a clean atmosphere reduces visual range by 30%. The addition of the same amount when the background visual range is 35 km (20 miles) produces a 3% reduction.

From a comparison of $PM_{2.5}$ and PM_{10} levels at Canadian rural and urban sites, a noticeable change in visual range could occur for a 1-2 µg/m^3 increase in PM_{10} at rural sites and a 2-5 µg/m^3 increase at urban sites. The deciview scale does not consider what the particle level is, rather, it considers the incremental change in visibility, or percent change in particulate matter, from existing levels.

In a recent study in the Lower Fraser Valley of British Columbia, judgements of acceptable or unacceptable visibility were made from photographs (Pryor et al., 1994b). Other studies have shown that there is a high correlation between judgements made from photographs and those made in field (Malm et al., 1981; Middleton et al., 1985). In a public perception study in Denver (Ely et al., 1991), acceptable visibility as measured by visual range was approximately 50 km. Acceptable visibility or acceptable changes in visibility in different regions of Canada still need to be determined.

The effects of changes in particle levels on visibility across Canada have also been evaluated from visual range estimates from meteorological synoptic observing stations located at airport sites (Stuart and Hoff, 1994). Trained observers using a series of visibility markers, for which the observer knows the distance, estimate airport visibility. Using a technique based on the frequency distributions of visual range, median visual range, from observations less than 24.1 km and estimated visual range when greater than 24.1 km, were computed for 140 stations. From this analysis, Ontario sites have lower visibility in the summer, reflecting higher $PM_{2.5}$ levels in the summer season. Consequently, measured summer visibility would be expected to be lower than those calculated from mean $PM_{2.5}$ levels (1984 to 1993). In Vancouver, $PM_{2.5}$ levels reach a maximum in winter, and consequently actual summer visual ranges would be greater than calculated from annual averages (1984 to 1993).

Contours of visual ranges were determined from the 140 stations across Canada. Median summer and winter visual ranges for 1951 to 1991, with relative humidity less than 80%, are shown in Figures 7.5 and 7.6. For many regions of Canada, summertime visual ranges are greater than 80 km. However, southeastern Canada is notable for significantly lower summertime visual ranges. Wintertime visibility is dramatically lower in the western parts of Canada compared to the summertime. Visibility across the country becomes uniform in winter.

These regional differences in visual range have also been observed from visibility measurements taken in Canada at non-urban sites. The visual range measured at Egbert (86-120 km) was lower than the visibility measured at Waterton (210-350 km) and St. Andrews (185-210 km) (Table 7.3). Visual ranges calculated for minimum and maximum mean $PM_{2.5}$ levels (average 1984 to 1993, see Table 7.4) at NAPS stations in Canada ranged from 40 to 140 km. The visual ranges are similar to those observed in the US during 1951 to 1991 (Malm, 1992; Malm et al., 1994a).

Figure 7.5 Contour Plot of Summer Visual Range (from Stuart and Hoff, 1994)

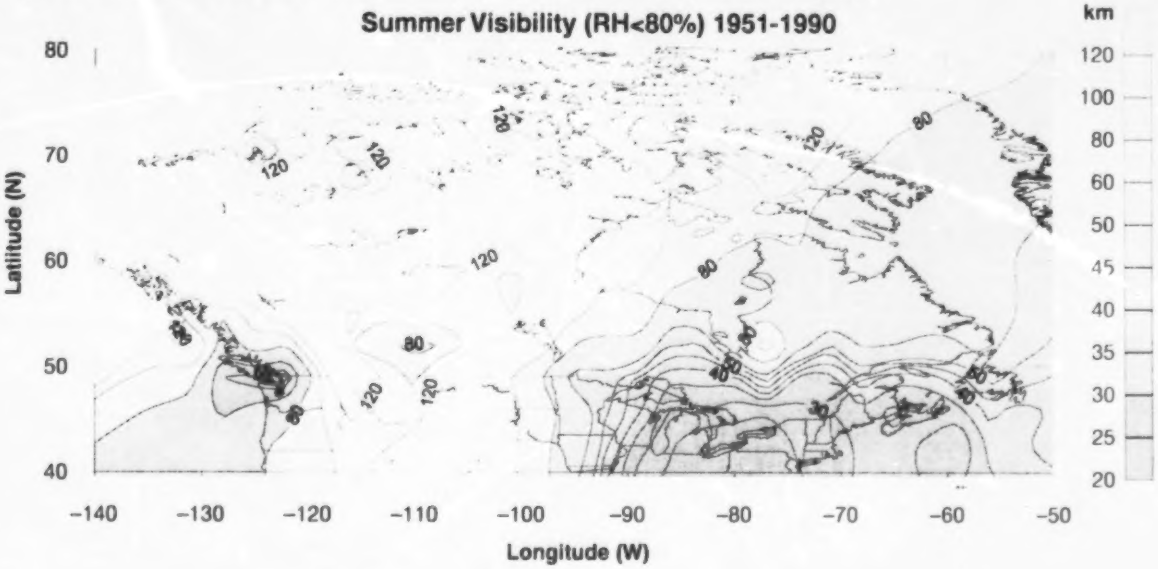
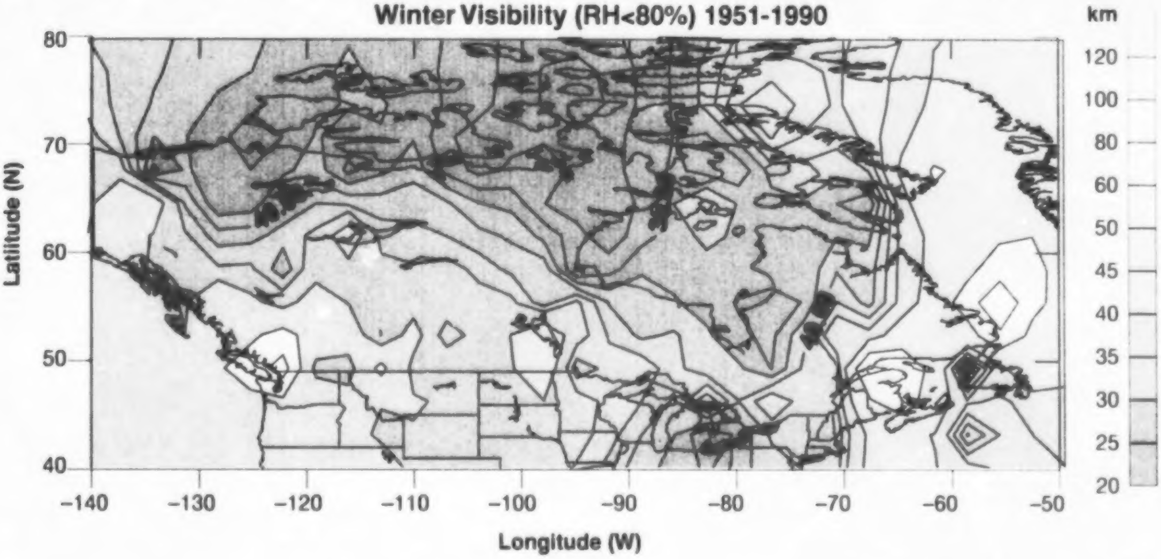


Figure 7.6 Contour Plot of Winter Visual Range (from Stuart and Hoff, 1994)



7.5 SUMMARY

Submicron particles, primarily those in the 0.3 to 0.7 μm size range, are the most effective in reducing visibility. There is empirical evidence showing that there is a linear relationship between the light scattering coefficient (b_{scat}) and fine particle mass, with b_{scat}/fine mass = 3.1 m^2/g. Thus, with the benefit of the Koschmeider equation and known relationships for b_{scat}/b_{ext}, visual range may be determined as function of PM_{10} mass concentrations if the ratio of $PM_{10}/PM_{2.5}$ is known, or directly from coarse and fine particle masses. However, it should be recognized that the fine fraction is primarily responsible for degraded visibility.

Natural visual range estimates in Canada, which are based on b_{scat} measurements at rural sites are in the range of 86 to 350 km depending on local relative humidity conditions. Southeastern Canada has the lowest estimated natural visual range of 86-120 km, which is similar to the visual ranges observed in the United States (100-180 km, Table 7.3). Western and eastern Canada have visual ranges of 210-350 km and 185-210 km respectively. These visual range estimates correspond with natural $PM_{2.5}$ levels of approximately 3.2 to 5.5 μg/m^3 in western Canada, 5.5–6.1 μg/m^3 in eastern Canada, and 10-13 μg/m^3 in southeastern Canada. These levels are consistent with other estimates of natural background levels of $PM_{2.5}$ for North America ranging from 1-5 μg/m^3 (see Chapter 5).

Visual range can be calculated using the Koschmieder equation and assuming that $b_{scat}/b_{ext} = 0.9$ for rural areas = 0.7 for urban areas, and $b_{scat}/PM_{2.5}$ = 3.1 m^2/g. Using a background $PM_{2.5}$ level of 5 μg/m^3 gives a natural visual range of 230 km for an unpolluted, rural atmosphere. There are regional differences. Regional natural background $PM_{2.5}$ levels, estimated from visual range measurements, range from 5.5 to 13 μg/m^3 (Table 7.3). A 10% increase in these values (i.e. ranging from 6 to 14 μg/m^3) would normally be visually detectable. Any increase in particle levels above these levels would reduce visual range. Conversely, a 10% decrease in fine particle concentrations (improvement) would also be visually detectable.

8 EFFECTS ON VEGETATION

The research community acknowledges the effect of suspended particles on vegetation in the research community, however, researchers have not focussed the same level of effort as has been devoted to the study of the effects of phytotoxic pollutants (NO_2, SO_2 and O_3) (Farmer, 1993). The high deposition of inert particles from the use of fertilizers and lime, and by the emissions of cement dust from cement works in the early 1900s prompted the first research on the effects of dust on vegetation (Pierce, 1909; Duggar and Cooley, 1914).

Stomata are the primary entry point of atmospheric pollutants into vegetation. Typical stomatal openings are 10-12 µm long by 2-8 µm wide (Smith, 1990; Farmer, 1993). Some corn and cereal stomata are up to 20-32 µm long and 8-12 µm wide (Krajickova and Mejstrik, 1984). The primary mechanisms by which particles affect vegetation (Pyatt and Haywood, 1989; McCune, 1991; Farmer, 1993) are by:

- physical smothering of the leaf surface,
- physical blocking of stomata through particle lodging or penetration of stomatal apertures,
- inducing a chemical effect due to particle composition, and
- indirect effects on soil pH and ionic composition.

There are three ways particles can deposit upon leaf surfaces: by sedimentation under the influence of gravity, by impaction under the influence of eddy currents and by deposition under the influence of precipitation (Bache, 1981; Smith, 1990). For particles of about 10 µm, impaction is efficient, and interception by fine hairs on vegetation is likely an effective retentive mechanism. The magnitude of the effect of the particles will be determined by the deposition rate under conditions of variable microclimates.

The physical smothering of the leaf surface causes reduced light transmission, affecting photosynthetic processes (Thompson et al., 1984; Pyatt and Haywood, 1989; Farmer, 1993). It is estimated that typical roadway depositions are <2 g dust/m^2, and the resultant effect of PM_{10} from automobile exhaust on the photosynthesis of trees is likely to be small (Thompson et al., 1984). Increases in the temperature of particle-covered leaves result in a positive impact on respiration and a negative impact on photosynthesis and productivity (Eller, 1977).

Chemical effects and metal accumulation have been observed from particle deposition. Sphagnum accumulated Al, Cr, Fe, Ni, Sc, Sm and V from a nearby gravel road with dust particles of 3-100 µm diameters (Santelmann and Gorham, 1988). Saline particles on vegetation caused accumulation of Cl, production of foliar lesions, and changes in levels of plant mineral nutrients and metabolites, physiological processes and growth and reproduction (McCune, 1991).

The physical obstruction of the stomata has been observed to reduce stomatal resistance, resulting in the potential for higher uptake of pollutant gases, and it may also affect the exchange of water vapour. There are several examples of this. In a deciduous forest, *Quercus petraea* (Mattuschka) Leibl (oak) leaves were studied for stomatal blockages (Williams et al., 1971). At mean deposition rates of 11.5 µg/cm^2 and 70.7 µg/cm^2, approximately 8% and 40%, respectively, of stomata were blocked open with particulate debris from a smokeless fuel plant. That same study also observed that accumulation of particles at a rate of 200–600 mg/m^2 interfered with normal stomatal behaviour by lowering stomatal resistance at night, and higher levels of total leaf sulphur were observed (Ricks and Williams, 1974). A study of laurel leaves near a steel works operation found that average daily smoke concentrations of 17–30 µg/m^3 caused approximately 50% blockage of stomata (Pyatt, 1973). In one instance, exposure to fly ash particles (83% were <8 µm) did not obstruct stomata, however, the particles did accumulate on the surface of guard cells, stimulating the mechanism regulating the opening and closing of the stomata and blocking the closing of the stomata (Krajickova and Mejstrik, 1984).

Particle accumulation on leaf surfaces may cause plants to become more susceptible to other stresses. Sassafras and grape leaves were observed to be more susceptible to leaf spot diseases caused by fungi than were leaves of comparable plants with no visible dust deposits; numbers of microflora were higher on dusty leaves; and second-year dusty hemlock needles were frequently more chlorotic (Manning, 1971).

Much literature describing the effect of cement dust on vegetation is available; however, it often fails to show the size distribution of the particles. Cement particle diameter ranges can be typically 1-100 μm (Hinds, 1982). It is estimated that 80-90% are less than 30 μm. Relatively high deposition rates (from 1 to 7 g/m^2 for up to 130 days) of cement dust on cereals have resulted in decreased respiration, catalase activity, oil content and overall yields (Borka, 1980; Singh and Rao, 1981; Shukla et al., 1990). Estimating whether the negative effects are governed by the sheer volume of particulate loading or by the size of the particles is difficult. A review of the effects of cement dust on trees showed that the dust caused physical damage to the leaves, reduced fruit setting and generally reduced growth (Farmer, 1993).

Small particles (10 μm) are of significance to forest ecosystems owing to long-range transport, preferential contamination with toxic metals and efficiency of vegetative capture via impaction (Smith, 1990).

Accumulation of metals and stomatal interference have been observed for particles in the 10 μm size range for a variety of vegetation types and particle sources. Insufficient information is available to estimate a relationship between ambient concentrations of particles less than 10 μm in diameter and vegetation effects. This relationship will be extremely complex, given the interactions between particle size, particle composition and vegetation species variability.

Conclusions

This review has focussed on the impact of inert particles and as such, describes the limited information available. Indirect and possibly significant effects on vegetation from: reduced light intensity (through particle loading of the atmosphere), secondary aerosols and nutrient imbalances through particle deposition to soils are not addressed. Neither are the potentially significant impacts of particles that are of similar or smaller size than the stomatal openings.

Conclusive information about the effects of PM on vegetation is quite limited and robust quantitative dose-response information is lacking.

9 EFFECTS ON MATERIALS

The deposition of particulate matter on materials can reduce their aesthetic appeal, increase their rate of physical and chemical degradation. Particles can act as catalysts for the conversion of SO_2 and NO_x to sulphuric acid and nitric acid (Santachiara et al., 1989). These acidified particles can accelerate the chemical degradation of the susceptible material surface on which they are deposited.

Most available information in the literature is on the effects of particle exposure in combination with SO_2. The following discussion will, however, focuses on the effects associated only with particulate matter exposure. Two categories of effects are discussed: 1) corrosion and erosion, and 2) soiling and discolouration.

9.1 CORROSION AND EROSION

9.1.1 Metals

Most studies (e.g., Sanyal and Singhania, 1956; Yocom and Grappone, 1976) have linked the presence of particulate matter in the air to the enhanced speed of corrosion on metal surfaces. Goodwin et al., (1969) found that damage to steel, protected with a nylon screen and exposed to quartz particles, became substantial when the steel was exposed to particles of diameter 5 µm or larger. The corrosion of iron in an atmosphere of 0.15% by volume SO_2 and at varying relative humidity values between 55% and 95% was found to increase in samples with carbonaceous particulate matter (Skerry et al., 1988a, 1988b; Askey et al., 1993).

There are two different views on how particulate matter, together with SO_x, plays a role in the corrosion of metals. The first states that the catalytic species in particulate matter (e.g., ash) accelerate the oxidation of SO_x. Therefore, SO_x alone will not lead to an acceleration in corrosion (Walton et al., 1982). The second view states that PM increases the conductivity of the metal surface moisture layer after the dissolution of soluble anions contained within the particle. The net effect is to enhance corrosion. The increase in atmospheric corrosion rates in this case is predominantly due to the quantity of species that can be leached from the particulate. If the leachate is of higher pH or contains significant quantities of inhibitive ions (e.g., Ca^{2+}), then the corrosion rate may not increase significantly (Askey et al., 1993).

9.1.2 Paint

Existing evidence shows that paint durability is altered by the presence of particulate matter (National Research Council, 1979). Some studies also suggest that more corrosive pollutants are carried on the particulate matter, allowing them to reach the underlying surfaces or to serve as concentration sites for other pollutants on painted surfaces (Cowling and Roberts, 1954). General Motors conducted field tests to study the effect of particulate matter on finishes of automobiles. The tests concluded that damage on the paint surface was mostly due to the reaction of calcium from dust with sulphuric acid in rain or dew, forming calcium sulphate deposits on the surface of the paint. Damage increased with increased exposure time; however, washing the panels within 24 hours of a wetting event significantly reduced the damage (Wolff et al., 1990).

9.1.3 Stone

Heavy metals contained in dust convert SO_2 to sulphuric acid and could thus accelerate stone corrosion (Luckat, 1972). Slow erosion of stone surfaces, similar to sandblasting, is also possible under strong wind conditions (Yocom and Upham, 1977).

The surfaces of buildings contain zones protected from rainfall and surface runs. With time, a hard, irregular, black crust usually covers these zones. This crust was composed of aerosols and particulate matter of atmospheric origin and is thought to play a role in the deterioration of the stone surface. Del Monte et al., (1981) plotted the particulate size distribution of a thin section of crust from northern Italy; they found the median number diameter to be very close to 10 µm. Following a series of analyses, the authors concluded that oilfired boilers played a substantial role in the formation of this crust. Generally, the crust composition will vary from site to site depending on the composition of the particulate airborne pollutants in the area (Del Monte et al., 1981; Sabbioni and Zappia, 1992).

9.1.4 Electronics

Particulate matter of natural and anthropogenic origin, ranging in size from tens of angstroms to 1 µm, can cause corrosion and failure of electronics. Failure can be caused by several factors, including the hygroscopic property of the particles or the formation of a conductive moisture film on insulating surfaces, thus leading to electrical leakage. Sinclair (1992) demonstrated from data collected in the 1980s that the indoor concentration of anthropogenic particles and their rate of arrival at surfaces is comparable to those of many corrosive gases in urban environments.

9.2 SOILING AND DISCOLOURATION

Soiling of materials is a concern, because of its potential aesthetic impact and because it precipitates more frequent cleaning and repainting, thereby reducing its lifetime usefulness and increasing costs associated with maintenance of the material. Haynie and Lemmons (1990) describe soiling as the contrast in reflectance of particles on a substrate to the reflectance of the bare substrate. Deposition studies have shown that more than 50% of the deposited matter could be due to particulates greater than 10 µm in diameter. However, it does not follow that these particulates are the major source of soiling, as it is known that fine particulates have greater light extinction characteristics (see chapter 7) and may be bound more strongly to surfaces.

Soiling also refers to degradation that can be remedied by cleaning, washing and/or repainting. However, determining the level of particulate matter accumulation that leads to increased cleaning is difficult. In a study by Hancock et al. (1976), visually perceived dust accumulation and a telephone survey were used to draw the conclusion that a dustfall rate of less than 0.17% surface coverage (effective area coverage) would be tolerated by the general public. Hamilton and Mansfield (1993) state that cleaning and repainting are usually triggered following a 30% loss of reflectance.

9.2.1 Building Materials

Theoretical dose-response relationships have been developed with the intent to describe the soiling of surfaces by airborne particles and predict the necessary cleaning frequencies. For example, Beloin and Haynie (1975) conducted experiments in five locations in the US over a two year period. Six different building materials were tested: painted wood, concrete block, brick, limestone, asphalt shingles and window glass. TSP levels among the sites ranged from 60-250 mg/m^3. Surface reflectance was used as a measure of soiling. The authors found that, except for asphalt, soiling of all the material was directly proportional to the square root of the TSP dose, with TSP dose equal to the product of TSP level and exposure time. For asphalt, soiling was directly proportional to the TSP dose. Van Aalst (1986), in a review of the literature, stated that the major fault in models presented to date was the lack of experimental verification.

Hamilton and Mansfield (1991,1993) attempted to address this issue, focussing their work on white-painted wood. A number of equations were presented for the calculation of reflectance of a surface based on experimental data and for the calculation of soiling rate based on models. These theoretical models describe soiling as a function of either TSP (Haynie, 1986) or elemental carbon (EC) (Lanting, 1986). The conclusion drawn was that for model output and experimental results to agree, many factors must be considered in choosing the right equations to apply. Two factors appear to be especially critical:

1) The physical and chemical composition of the airborne particles, especially the proportion of EC, since their strong light absorption properties will affect the soiling rate. Recent increases in the proportion of fine particles and EC (from diesel emissions primarily) in ambient PM requires some modifications to the model equations which were derived at a time when coarse particles comprised a larger fraction of PM and when emissions were predominantly from coal combustion.
2) Whether or not the surface is sheltered from the rain, which if not protected, will promote cleaning, by washing PM (primarily coarse particles) off the surface of the material. Exposed surfaces, therefore, tend to have slower soiling rates than sheltered surfaces although the experimental data demonstrated that the effect of rainfall on soiling has some complexities to it (e.g., rainfall may lead to redistribution of particles on surfaces with a consequent increase in localized soiling).

Table 9.1 summarizes some of the experimental data presented in Hamilton and Mansfield (1993). Soiling

Table 9.1 A Summary of Soiling Rates for White-painted Wood in a Tunnel and an Ambient Environment (Hamilton and Mansfield, 1993)

Exposure time	% loss in reflectance		
	Road tunnel [C_{tsp} = 208 μg/m^3]	Ambient atmosphere [C_{tsp} = 45 μg/m^3]	
		Sheltered wood	Exposed wood
50 days	25	11	9
100 days	45	18	15

was greater in both of the sheltered environments than in the exposed situation; however, the five-fold difference in TSP levels between the two experimental sites was not matched by a proportional change in reflectance (the latter changed by a factor of two to three). The authors report that the difference in observed reflectance is consistent with soiling being proportional to the square root of TSP dose, as described above, for example, by Beloin and Haynie (1975).

Haynie and Lemmons (1990) also conducted a soiling study aimed at determining the environmental factors that play a role in the soiling of white-painted surfaces. Five conclusions were reached by these authors:

- Coarse particles (between 2.5 and 10 μm) initially contribute more to soiling of both horizontal and vertical surfaces than fine particles (≤2.5 μm).
- Coarse-mode particles, however, are more easily removed by rain than are fine particles.
- For sheltered surfaces, reflectance changes are proportional to surface coverage by particles, and particle accumulation is consistent with deposition theory.
- Rain interacts with particles to contribute to soiling by dissolving particles and leaving stains.
- Very long-term remedial actions are probably taken because of the accumulation of fine rather than coarse particles.

Similar results were also reported by Creighton et al. (1990).

9.2.2 Fabrics

There is no recent information in the published literature on the effect of particulate matter on fabrics. Earlier studies show that abrasive particles are damaging to fabrics. Also, the soiling of fabrics by particulate matter increases washing frequency, thereby reducing their life usefulness.

9.2.3 Indoor Soiling of Works of Art

Potential soiling of artwork in five southern California museums was studied by Ligocki et al. (1993). Buildings with sophisticated ventilation systems and particle filters showed much lower seasonally averaged concentrations of particulate matter. For example, museums with good ventilation/filter systems typically had indoor fine particulate concentrations of less than 10 μg/m^3, whereas one museum with no control system had indoor fine particulate concentrations averaging 60 μg/m^3. The authors found fine elemental carbon and soil dust particles from the outdoor environment present indoors.

Nazaroff and Cass (1991) proposed several means by which to reduce the hazard of art soiling, including reducing the building ventilation rate, increasing the effectiveness of particle filtration systems, the use of display cases and the elimination of indoor particle sources. The authors predicted that such control measures could reduce the soiling rate by at least two orders of magnitude.

9.3 CONCLUSIONS

The limited amount of scientific literature supports observational data that particles affect materials either by soiling, eroding or corroding the surface. Specific information on the effects of chemical composition and quantitative dose-response relationships are not currently available, nor is it currently possible to quantify the particle exposure level at which materials damage and soiling are perceived. It should be noted, however, that the soiling potential of fine particles is greater than that of coarse particles, given the greater rate of light extinction.

10 TOXICOLOGY

Toxicological studies involve the controlled testing of experimental animals (or tissue samples) to known quantities of PM under laboratory conditions. Whereas the human epidemiological studies discussed in Chapter 12 reflect exposure to the full range of ambient particle mixtures, the studies with experimental animals have, for the most part, been restricted to well-defined particle species. Although not directly comparable to the complex ambient particle mix, the animal toxicology studies provide valuable information about mechanistic aspects of particle toxicity, including the fate of particles in the lower respiratory tract, and information describing the biological responses that particles can elicit in the lung. In toxicological studies, PM are administered either by inhalation, or by intratracheal instillation.

Inhalation studies themselves can be conducted as whole-body exposures or as nose/mouth-only exposures. Generally, the interpretation of whole-body exposure studies can be complicated by the systemic effects of ingestion of the test substance during preening. Nevertheless, for the most part, the effects of concern observed in the published studies are limited to the airway itself, minimizing any potential confounding effects of ingestion.

Particle size is believed to be the most important characteristic influencing deposition in the respiratory system (Lippmann, 1977; Dockery and Pope, III 1994; Anderson et al., 1990). Since fine particles ($PM_{2.5}$) readily enter the respiratory system they are considered to pose a greater health risk than coarse particles. Up to 80% of PM_{10} and 60% of the total suspended particles (TSP) are made up of $PM_{2.5}$. Therefore, in this review we have paid great attention to particle sizes used for animal toxicology studies.

It should be noted that for the purposes of this review, the animal toxicology assessment will be limited to the particle effects *per se*. Where individual chemical compounds are identified on particles, the toxicology assessment will be limited to the chemical's effect as an integral part of the particle, except where the toxicity of the identified chemical is central to the toxicity of the particle.

It should also be noted that considerable uncertainty is involved in extrapolating results from animal inhalation studies and applying these results to humans for purposes of quantitative risk assessment. These uncertainties relate to differences in the sensitivities of specific target cells, in cell populations in the individual airway generations of animal species, in metabolic activities of lung cells, in the lifespans between laboratory animals and humans, and also in the dosimetry in the respiratory tract. The issue of animal to human extrapolation is addressed in Section 10.10.

This review on animal toxicology studies is a summary based extensively upon a contract report prepared for Health Canada, entitled "Particulate Air Pollution: Animal Toxicology" (Oberdörster et al., 1994b). Recently, a large body of literature on particle toxicity studies have come to light, providing further evidence concerning the impact of particles on experimental animals. In particular, data from several animal studies have shed light on the potential underlying mechanism of particle-induced cardiovascular diseases. This may provide biological plausibility for the recent epidemiological findings of a consistent association between fine particle air pollution and increased hospital admissions due to cardiovascular diseases (Burnett, 1995; Schwartz and Morris, 1995). Thus, in this report a great effort is made to provide the latest available data, in an attempt to assess the mechanisms and concentrations of particle-induced toxicity.

The studies reviewed in this report are divided into those involving acute and short-term exposure, and those that used subchronic and chronic exposure. An acute exposure is defined as single 4 or 6 h exposure to the test chemical. A subchronic study generally involves repeated exposure for approximately one tenth of the lifetime of the species studied. Chronic studies usually mimic those encountered in the workplace with animals exposed 6 h per day, 5 days per week for their lifetime.The effects of PM on a number of different health endpoints are considered: lung function, host defences, tissue cellular and biochemical responses, mortality, cardiovascular response, effects on compromised lungs and cancer.

The effect of combined exposures of PM with other air pollutants is also considered. The chapter concludes with a discussion of the critical issue of animal to human extrapolation, and provides a discussion of the mechanistic aspects of particle-induced health effects.

10.1 VENTILATORY LUNG FUNCTION

10.1.1 Acute and Short-Term Exposure

In some widespread geographic areas of North America, silica can be found among important ambient PM constituents. Studies involving intratracheal instillation of silica (<5 µm) in rats demonstrated changes consistent with airflow obstruction, with a restrictive lung disease component, although the amounts of silica used (10-40 mg/animal) were well beyond those likely to be encountered in the environment (Gross et al., 1984; Wright et al., 1988; Churg et al., 1989). Similar levels of iron oxide (0.1 µm MMAD, 10 mg) showed no significant changes in a variety of pulmonary function parameters (Wright et al., 1988). The instillation of 100 mg latex microspheres (0.1 µm) into the lobes of sheep also failed to demonstrate any altered pulmonary function (Begin et al., 1985).

Guinea pigs exposed for 1 h to aerosols of sodium sulphite (0.36 µm MMAD, 474–972 mg/m^3) showed dose-related decreases in total lung capacity, vital capacity, functional residual capacity, residual volume and diffusion capacity (Chen et al., 1987).

The role of particle acidity in potentiating toxicity has been demonstrated in a series of studies. Inhalation of zinc oxide particles (0.05 µm, 0.87 mg/m^3) coated with sulphuric acid at a concentration of 20 µg/m^3 resulted in hypersensitivity responses of the conducting airways of guinea pigs, whereas the zinc oxide without the acid coating did not produce such effects. A 10-fold higher concentration of acid alone was required to produce similar effects (Chen et al., 1992a). A 1 h exposure to zinc oxide alone at 6 mg/m^3 also showed no effects (Chen et al., 1991a). Similar exposures to acid-coated copper oxide particles (<0.1 µm, 1-2 mg/m^3) caused a prolonged change in pulmonary mechanics (Chen et al., 1991b).

Fine H_2SO_4 aerosols have been shown to induce nonspecific airway hyper-responsiveness to stimuli, a hallmark of human asthma, in otherwise healthy animals. El-Fawal and Schlesinger (1994) exposed rabbits for three hours to submicrometre sulphuric acid aerosols (50–500 µg/m^3) and then used an *in vitro* assay to determine the airway responsiveness to acetylcholine or histamine. Hyper-responsiveness was observed following acid aerosol exposure concentrations >75 µg/m^3 (El-Fawal and Schlesinger, 1994). When rabbits were exposed to 250 µg/m^3 of H_2SO_4 for 1 hour per day, 5 days per week for 4 months, H_2SO_4 caused bronchial hyper responsiveness and alterations in airway diameter and secretory cell number, while pulmonary mechanics parameters did not show significant change (Gearhart and Schlesinger, 1989). The underlying mechanism for hyper-responsiveness appears to be interference with normal contractile/dilatory homeostatic processes in the airways via modulation of airway receptors involved in maintenance of airway tone. This assumption is supported by an observation of decreases in the production of pulmonary prostaglandins E_2 and $F_{2\alpha}$, and thromboxane B_2, by H_2SO_4 inhalation (250-1000 µg/m^3, 1 h/d for 5 days) in rabbits (Schlesinger et al., 1990). These eicosanoids serve as potent mediators of smooth muscle tone and inflammatory response.

When guinea pigs were exposed to fly ash (0.21 µm MMAD, ~6 mg/m^3) from low-acid Montana lignite coal or high-acid Illinois no. 6, only those exposed to the high-acid coal showed reduced pulmonary function, which persisted for up to 96 h post-exposure (Chen et al., 1990).

A possible role of endotoxin contamination of particles, which may be a component of indoor air pollution, as a cause of particle-associated changes in ventilatory function is suggested in the study by Gordon (1992). Guinea pigs exposed to machine oil aerosols (10-100 mg/m^3) contaminated with endotoxin (0.3-5.3 µg/m^3) showed significant decreased airway conductance, whereas non-contaminated oils produced no effects.

10.1.2 Subchronic and Chronic Exposure

Most subchronic and chronic exposure studies in which the ventilatory status of animals was measured involved multiple instillations of relatively high mass burdens of particles or aerosol exposures performed at mass concentrations >1 mg/m^3. For essentially all types of particles investigated to date, such exposures have been found to cause significant compromises in various lung ventilatory functions. The severity of changes observed with a given particle type generally increases with exposure duration and mass concentration.

Instillation of 100 mg of silica into the lungs of sheep every 10 days for 18 months resulted in decreased pulmonary functional status, including decreased lung compliance, vital capacity and diffusion capacity (Begin et al., 1989).

Exposure to diesel exhaust particles resulted in increased pulmonary flow resistance in guinea pigs (~6 mg/m^3, 20 h/d, 7 d/wk) for 28 days (Wiester et al., 1980). Fedan et al. (1985) showed that the contractile response to acetylcholine was increased by chronic, two-year exposure of rats (7 h/d, 5 d/wk) to diesel exhaust (2 mg/m^3), coal dust (2 mg/m^3) or a combination of the two (additive response). Mauderly et al. (1988) exposed rats for up to 30 months (7 h/d, 5 d/wk) to diesel exhaust at 0.35, 3.5 or 7.1 mg/m^3 and reported reduced lung volume and compliance at the higher doses.

Pulmonary function changes were reported in rats following two-year exposures (6 h/d, 5 d/wk) to silica (1 mg/m^3) or test toner (16 mg/m^3). However, no effects were seen after exposure to titanium dioxide (5 mg/m^3) (Heinrich et al., 1989). Guinea pigs showed a decreased tidal volume throughout a one-year chronic exposure to cotton dust (21 mg/m^3, 6 h/d, 5 d/wk) (Ellakkani et al., 1987). In rats exposed to aerosolized volcanic ash (1.6-2.5 μm MMAD, 5-50 mg/m^3) or quartz (1.7-2.5 μm MMAD, 50 mg/m^3) for up to 12 months (6 h/d, 5 d/wk), only the quartz-exposed lungs developed significantly higher lung volumes, and both quartz-exposed animals and those exposed to the highest concentration of volcanic ash studied had elevated respiratory rates (Wehner et al., 1983).

Rabbits exposed to submicrometre sulphuric acid aerosols delivered at a mass concentration of 250 μg/m^3 for 1 h/d over a period of four months showed an increase in airway sensitivity to acetylcholine (Gearhart and Schlesinger, 1986). The cumulative dose inhaled by the rabbits is similar to current peak daily doses from ambient exposure in some areas of North America.

Unfortunately, the studies done to date do not provide any insight into how different sizes of the same type of particle may affect ventilatory function, and it generally remains unknown how subchronic or chronic exposures in the range of ambient concentrations might affect lung function.

10.2 HOST DEFENCES

10.2.1 Mucociliary Clearance and Conducting Airways

Acute and Short-Term Exposure

The main lines of defence against particles that deposit in the conducting airways are a mucous lining that entraps deposited particles and a cilia-based mucociliary transport system that conveys mucus and associated constituents out of the lower respiratory tract. Acute exposures to a variety of types of particles have been shown to affect mucociliary clearance or the action of cilia that propel airway mucus. The exposure of hamsters for 2 h to nickel chloride (<1 μm) at a mass concentration of 100 μg/m^3, for example, has been shown to decrease ciliary beat frequency (Adalis et al., 1978). On the other hand, Schiff et al. (1979) found no consistent changes in ciliary beat frequency after hamsters were exposed for 3 h to carbon black (0.3 μm) at a mass concentration of 1.5 mg/m^3. A 5 d inhalation exposure to volcanic ash (0.65 μm MMAD, 9.4 mg/m^3) showed no effects in hamsters (Grose et al., 1985). Such findings suggest that changes in ciliary beat frequency following particle deposition in the airways are a function of the chemical characteristics of the particles.

Differing effects on mucociliary clearance have been observed with acid aerosols. Two studies performed with rabbits (Chen and Schlesinger, 1983; Naumann and Schlesinger, 1986) showed that 1 h exposures to submicrometre-sized sulphuric acid enhance mucociliary clearance of inert tracer particles at <1 mg/m^3 but decrease clearance at higher doses. Exposures of mice to 3.2 μm sulphuric acid droplets (15 mg/m^3) reduced mucociliary clearance, whereas exposure to 0.6 μm sulphuric acid particles (1.5 mg/m^3) had no effect (Fairchild et al., 1975). In the rat, a 4 h exposure to sulphuric acid (0.4 μm) at a mass concentration of 3.6 mg/m^3 caused no changes in short-term clearance (Phalen et al., 1980). In hamsters, however, a 2 h exposure to submicrometre-sized sulphuric acid at a mass concentration of ~860 μg/m^3 resulted in a reduction of ciliary beat frequency, which presumably would translate into a reduction in mucociliary clearance. The collective findings made in these studies suggest that lower doses, as a function of both concentration and time (C × T), increase mucociliary clearance, whereas higher doses have the opposite effect. These studies also show that the dose-response relationship for this effect is species-dependent.

Subchronic and Chronic Exposure

Relatively few studies have been performed to examine how subchronic or chronic exposure to particles may affect the airways and mucociliary clearance. In the guinea pig, four-week exposures to sulphuric acid aerosol at a mass concentration of 1 mg/m^3 resulted in airway epithelial injury and the loss of cilia (Nagai et al., 1991). In donkeys, six-month exposures to submicrometre-sized sulphuric acid at a mass concentration of 104 µg/m^3 resulted in a slowing of bronchial clearance (Schlesinger et al., 1979). A four-month exposure of rabbit to H_2SO_4 (250 µg/m^3) also resulted in a reduction of mucociliary clearance (Gearhart and Schlesinger, 1989). Rabbits exposed to sulphuric acid aerosol at 125 µg/m^3 (0.3 µm MMAD) for up to one year showed progressive slowing in clearance times (Schlesinger et al., 1992a). An increase in the numbers of secretory cells in the small airways at 12 months was also shown.

The slowing of tracheobronchial mucociliary clearance reflects changes in mucus secretory capacity, alteration in the type of mucus produced, and disruption of normal morphology of cilia and ciliated cells, which, again, can lead to the impairment of pulmonary defense capacity (Samet and Cheng, 1994).

10.2.2 Pulmonary Macrophage Response and Function

Acute and Short-Term Exposure

The alveolar macrophage response is the primary, first-line defence of the alveolar region against inhaled particles. Particle deposition in the alveoli can cause a relatively prompt expansion in the size of the alveolar macrophage population and also an influx of polymorphonuclear leukocytes (PMN) onto the alveolar surface. In the rats that had received intratracheal instillations of polystyrene microspheres (1.9 µm), Lehnert et al. (1985) found that the free cell population size increased nearly five-fold as of one day after the instillations. Although the PMN response had subsided as of day 14 after the instillations, the alveolar macrophage population remained persistently elevated at least 30 d after particle deposition.

The importance of particle size and particle numbers in determining the magnitude of the response has been shown using intratracheal instillation studies with latex microspheres (0.1 and 1.0 µm) and carbon (0.03 µm) in mice (Adamson and Bowden, 1981) and with carbon particles and coal dust in hamsters and rats (Brain and Cockery, 1977). In all cases, the smaller particle size (or greater number) led to a greater increase in the numbers of alveolar macrophages and PMNs (with equivalent dose mass). Also, more phagocytes are recruited with increasing lung burden (Brain and Cockery, 1977). Particle size also affects phagocytosis. In mice, maximal phagocytosis of polystyrene and phenylated microspheres took place in the size range of 1.0-2.0 µm, and particles with hydrophobic surfaces were more readily phagocytosed by peritoneal macrophages (Tabata and Ikada, 1988).

Some particles, however, decrease alveolar macrophage numbers, possibly through their cytotoxic effects — e.g., silica (Civil and Heppelston, 1979), cadmium chloride (Koshi et al., 1978) and manganese dioxide (Adkins et al., 1980).

Particles can also affect the phagocytic activities of alveolar macrophages. In mice exposed for 2 h to fly ash or silica (~1.6 µm MMAD, ~22 mg/m^3) decreased alveolar macrophage activity was seen *in vitro* (Fisher and Wilson, 1980). Similar results were seen for alveolar macrophages from rats exposed to quartz (10-100 mg/m^3) for 6 h or 3 d (Warheit et al., 1991). Conversely, rats exposed to iron oxide for 2 h (1.6 µm MMAD, ~22 mg/m^3; Lehnert and Morrow, 1985) or 3 h (0.15 µm MMAD, 271 mg/m^3; Kavet et al., 1987) showed increased alveolar macrophage activity *in vitro*. Rabbits exposed to sulphuric acid aerosol (0.3 µm, 0.5 mg/m^3) showed decreased mobility of alveolar macrophages after 7 or 14 d of exposure (Schlesinger, 1987).

To determine if particle size influences toxic effects of H_2SO_4, Chen et al. (1992) examined the effects of fine (0.3 µm) and ultrafine (0.04 µm) H_2SO_4 aerosols (300 µg/m^3) on biochemical and cellular parameters of bronchoalveolar lavage fluid from exposed guinea pigs. Four days of exposure (3 h/d) to fine aerosols enhanced the phagocytic activity of recovered alveolar macrophages, while an identical exposure to ultrafine aerosols depressed phagocytic function. A reduction in intracellular pH was noted 24 h following 4 d of exposure to the ultrafine aerosols, but not the fine aerosols. Thus, acid exposure produced a change in intracellular pH and phagocytic function of the alveolar macrophages, and the effect was particle size-dependent. Since the same mass concentration of H_2SO_4 was used for aerosols of both sizes, ultrafine H_2SO_4 aerosols as a population may have delivered a higher dose to the macrophages than fine H_2SO_4 aerosols. Enhancement of phagocytosis at a low dose and depression of phagocytosis at a higher dose have been reported in macrophages exposed to acidic media *in vitro* (Schlesinger et al., 1990). Therefore, the differences in response between fine and ultrafine aerosols may be attributable, to some extent, to the doses of acid aerosols delivered to macrophages *in vivo*.

Although the increase in lung phagocytes provides more cells to ingest particles, these same cells can also be the cause of injurious responses via the production of reactive oxygen species, hydrolytic enzymes, arachidonic acid metabolites and pro-inflammatory/pro-mitogenic cytokines.

Nadeau et al., conducted a series of *in vitro* studies (Nadeau et al., 1995; Nadeau et al., 1996) using human and rat alveolar macrophages obtained by lung lavage. Although a low dose of urban particles (up to 100 µg) induced an oxidative burst (Nadeau et al., 1996), the major effect was the suppression of phagocytosis of rat alveolar macrophages. More significantly, pre-exposure of human or rat macrophages to urban particles suppressed respiratory burst upon secondary challenge with zymosan, a stimulus (Nadeau et al., 1995). This reveals an impairment of bactericidal function.

Recently (Costa et al., 1994), the pulmonary inflammatory response and acetylcholine-induced bronchoconstriction in rats instilled with Mount St. Helen's dust, ambient particles from Düsseldorf or residual fly ash (all 2.5 mg) were suggested to be related to surface-coordinated Fe^{3+} and its role in generating oxidants in the lung. Ghio et al. (1992), using three types of silica dust with differing levels of surface-complexed Fe^{3+}, have also demonstrated that the inflammatory response in rats increased with increased Fe^{3+} levels.

Subchronic and Chronic Exposure

Subchronic and chronic exposures to many types of particles result in an accumulation of alveolar macrophages in the alveoli. Examples include diesel exhaust (six and 12 months, 250–6000 µg/m^3; Strom, 1984) to rats; carbon black (six weeks, 0.24 µm MMAD, 7 mg/m^3; Strom et al., 1989) to rats; cobaltous chloride (16 weeks, 1 µm MMAD, 2 mg/m^3; Johansson et al., 1986) to rabbits; and titanium dioxide (two years, 1.6 µm MMAD, 10-250 mg/m^3; Lee et al., 1985a) to rats. However, cytotoxic particles — e.g. nickel oxide (four weeks and four months, 0.4 µm MMAD, 15-800 µg/m^3 to rats) — can cause reductions in alveolar macrophage numbers (Spiegelberg et al., 1984).

Concurrent with the increases in alveolar macrophages observed with high lung burdens of poorly soluble particles are reductions in lung clearance rates, which indicate compromises in alveolar macrophage-mediated alveolar clearance. Rats exposed to coal fly ash (3 µm MMAD, 10.4 µg/m^3) for one month showed very little lung clearance during the 10 month post-exposure period (Matsuno et al., 1986). Similar results have been shown in 24 month studies with test toner (4 µm MMAD, 1-16 mg/m^3), titanium dioxide (1.1 µm MMAD, 5 mg/m^3) and silicon dioxide (1.4 µm MMAD, 1 mg/m^3) (Bellmann et al., 1991). This effect has become known as "particle overload." Both theoretical and experimental evidence indicate that the translocation of alveolar macrophages from the lung ceases when these phagocytes contain burdens of particles that are equivalent to ~60% of their original volume (Morrow and Mermelstein, 1988; Lehnert, 1990; Oberdörster et al., 1992b). However, such overload conditions have occurred only after chronic exposure of experimental animals to inhaled concentrations of several milligrams per cubic metre and are not expected to occur at ambient concentrations of <200 µg/m^3.

Kleinman et al. (1995) investigated exposure of rats for eight weeks to two important constituents of the fine particle fraction of PM_{10}, sulphate (0.2 µm MMAD, 70 µg/m^3) and ammonium nitrate (0.6 µm MMAD, 350 µg/m^3), and to the coarse mode of ambient aerosol (>2.5 µm MMAD) using resuspended road dust (4.0 µm MMAD, 300 and 900 µg/m^3). Decreases in alveolar macrophage function (except low-dose road dust) and increased lung permeability (only for nitrate and sulphate) were reported. Histopathological changes in the alveolar space were moderately changed in the order nitrate > sulphate > road dust. It appears that the fine fraction of PM_{10} was more toxic than the coarse fraction.

10.2.3 Antimicrobial Defence and Immunological Function

Acute and Short-Term Exposure

The acute deposition of particles in the lung can cause alterations in the lung's defence mechanisms against microbial infections; however, these effects appear to be related to the physicochemical characteristics, including acidity, of the particles deposited and not simply due to a particle effect.

The intratracheal instillation of 100 µg of urban air particles (0.4 µm) and coal fly ash particles (0.9 µm) into the lungs of mice was found to moderately enhance susceptibility to bacterial infection, whereas diesel particles (0.6 µm), silica (<5 µm) and volcanic ash (1.4 µm) caused only small increases in infectivity (Hatch et al., 1985). Even brief aerosol exposures to acid particles delivered at relatively low mass concentrations can affect mechanisms that would be expected to alter lung defence against microbes. This conclusion is illustrated in the study by Zelikoff and Schlesinger (1992) in which production of tumour necrosis factor-α (TNF-α) and superoxide anion by rabbit alveolar macrophages was decreased after the animals were exposed for 2 h to 50-125 µg/m^3 sulphuric acid (0.3 µm). On the other hand, exposure of mice for 4 h/d for four days to carbon black at a mass concentration of 10 mg/m^3 (Jakab, 1993) did not increase the animals' susceptibility to experimentally imposed infection.

Some investigations have shown that when the particle size is small enough (MMAD <0.05 µm), even the particles with little intrinsic toxicity can become very toxic. For example, Oberdörster et al. (1992) performed intratracheal instillation of fine (>0.2 µm) or ultrafine (~0.02 µm) titanium dioxide particles (both 500 µg), which are highly insoluble particles of low intrinsic toxicity. The ultrafine particles induced substantially more severe acute inflammatory reaction, than the fine particles, as determined by an increase in protein leakage and the number of inflammatory cells in lung lavage fluid. The inflammatory response due to ultrafine particles was dose-dependent in the range of 65 to 500 µg. The authors also found that inhaled ultrafine titanium dioxide translocated into pulmonary interstitium via endocytosis by pulmonary Type I and Type II cells (Ferin et al., 1992; Lehnert et al., 1993), to a greater extent than fine titanium dioxide particles (Oberdörster et al., 1992; Ferin and Oberdörster 1992), was retained in the lung for longer periods (Lehnert et al., 1993; Oberdörster et al., 1994), and induced more severe pulmonary inflammation (Oberdörster et al., 1994). Oberdörster and co-workers, in recent data, have shown that freshly generated thermodegradation products of polytetrafluoroethylene (PTFE) containing singlet ultrafine particles (MMAD 26 nm) at concentrations as low as about 9 µg/m^3 inhaled for 30 minutes caused pulmonary inflammation in rats, along with the up-regulation of several inflammatory mediators including interleukin-6 (Oberdörster, 1995). The evidence that the surface area of the retained particles in pulmonary interstitium, rather than their mass, correlates best with the inflammatory response (Oberdörster et al., 1994). This implies that the induction of mediators (e.g., chemotactic factors, growth factors, enzymes) from macrophages is a function of the particle surface area that interacts with the receptors of alveolar macrophages.

Evidence exists that ultrafine H_2SO_4 aerosols (0.04 µm) result in greater decreases in both intracellular pH and phagocytic function of alveolar macrophages than do fine H_2SO_4 aerosols (0.27 µm) in guinea pigs (Chen et al., 1992). H_2SO_4-coated ultrafine zinc oxide particles have been shown to initiate changes in production of prostaglandin $F_{2\alpha}$ and leukotriene B_4 in guinea pigs (Chen et al., 1989), which are involved in mediating the tension of bronchial smooth muscle and inflammatory responses.

Subchronic and Chronic Exposure

Subchronic or chronic exposure to high mass concentrations of even relatively "benign" dusts can result in increases in susceptibility to microbial infection. Bacterial clearance was found to be diminished in mice that were exposed to 20 mg/m^3 titanium dioxide (<1.98 µm) for 10 or 20 days. Whereas this effect was not observed in mice exposed to 2 mg/m^3 of this type of particle (Gilmour et al., 1989a, 1989b), it should be noted that species' differences evidently can play a role in the outcome of infectivity studies. Guinea pigs exposed to 23 mg/m^3 titanium dioxide (<1.98 µm) for 20 h/d for 20 days (Baskerville et al., 1988) showed no evidence of an enhanced increase in susceptibility to infection.

Kleinman et al. (1995) exposed rats to ammonium sulfate (20 or 70 µg/m^3, 0.2 µm MMAD), ammonium nitrate (90 or 350 µg/m^3, 0.6 µm MMAD), resuspended road dust (300 or 900 µg/m^3, 4 µm MMAD), or purified air, 4 h/d, 4 d/wk for 8 weeks. Exposure to sulfate and nitrate particles and road dust significantly depressed macrophage-dependent lung defence functions (antigen binding to Fc receptors and respiratory burst activity), compared to purified air controls.

Exposures to some types of particles can also result in alterations in immunological responses in the lung and regional lymph nodes. The humoral response in rats following nickel oxide exposure (0.4 μm MMAD) was significantly weakened at 100 and 200 $\mu g/m^3$ after four weeks and at 25 and 150 $\mu g/m^3$ after four months of exposure (Spiegelberg et al., 1984). With other types of particles — e.g., diesel soot (24 months, 7 and 3.5 mg/m^3, 0.25 μm MMAD) — alterations in immunological responses were observed only after chronic exposures (Bice et al., 1985). These results indicate that particles of metals, with known cytotoxic properties, affect the immune system to a significantly greater degree. Species differences in changes in the immunological status of the lung in response to particle deposition have not been studied systematically.

10.3 TISSUE, CELLULAR AND BIOCHEMICAL RESPONSES

10.3.1 Histopathological and Cytological Changes

Acute and Short-Term Exposure

Acute exposures to particles can result in a range of histological and cellular disturbances, although most of the reported studies have involved the exposure of animals to very high mass lung burdens or mass exposure concentrations. As previously described, Wright et al. (1988) found evidence that intratracheal instillation of 10 and 30 mg quartz caused increases in intra-alveolar space sizes (i.e., emphysema) and thickening of the most peripheral airways in rats that were not detected after the instillation of 10 mg iron oxide. These histopathological lesions are considered a model of pulmonary fibrogenic, restrictive lung disease and/or chronic obstructive pulmonary disease (COPD). Callis et al. (1985) found increased levels of lung hydroxyproline 10 months after intratracheal instillation of 5 mg of quartz in mice. An equivalent lung burden of latex microspheres (0.9 μm) failed to elicit a similar response.

Particle size and surface characteristics play important roles in mediating the fibrotic response of quartz. In a study using a range of quartz particle sizes (1, 5, 7.8 and 11.2 μm) deposited at equivalent particle surface areas, larger quartz crystals were found to cause a greater inflammatory and fibrogenic response than smaller crystals (Wiessner et al., 1989), and surface charge has been shown to affect the fibrogenicity of silica particles (Wiessner et al., 1990). It must be noted, however, that the toxicological studies performed with crystalline quartz were at far greater levels than those occurring in the urban atmosphere.

In rats that were intratracheally instilled with 40 mg of three different volcanic ash samples (0.5-1.5 μm count median diameter (CMD)) obtained from three geographical locations, pure crystalline quartz (1.5 mm CMD) or soil quartz (1.6 mm CMD). The quartz caused early granuloma formation, later fibroplasia and collagen deposition, widespread lipoproteinosis and fibrosis in the regional lymph nodes (Sanders et al., 1982). The volcanic ash caused an ill-defined inflammatory reaction, with a few rats showing granuloma formation, a limited fibrosis, moderate lipoproteinosis and lymph node enlargements with evidence of collagen formation. The pulmonary response to the soil particles was least intense and qualitatively similar to the response seen with the volcanic ash. The soil particles, however, did not cause lymph node enlargements.

There are indications that surface-complexed iron on particles is involved in pulmonary injury. Guilianelli et al. (1993) examined the role of iron in PM-related lung injury using chrysotile, nemalite, and hematite particles incubated with rabbit tracheal epithelium *in vitro* (50-100 $\mu g/cm^2$). Surface available iron was correlated both with the oxidizing potency of mineral particles, and with cytotoxicity, the expression of cytokeratin-13 and the formation of cross-linked envelopes of tracheal epithelial cells (two markers of squamous metaplasia), which could be blocked by deferoxiamine, an iron chelator.

To determine potencies of specific metals, Berg et al. (1993) examined different fractions of fly ash (12.5-1000 μg/ml, MMAD <4 μm) for their ability to stimulate bovine alveolar macrophages to secrete reactive oxygen species, namely superoxide anion and hydrogen peroxide which play a key role in macrophage bactericidal function. They noted that the release of hydrogen peroxide was correlated, in descending order, with the metal contents in the fly ash particles: iron>manganese>chromium>vanadium>arsenic.

Subchronic and Chronic Exposure

Nearly all subchronic and chronic exposure studies that have been reported to date describe pathological responses to insoluble particles, regardless of the types of particle involved, the exposure mass concentrations studied, the duration of exposure or the species examined (although most studies have been conducted at particle concentrations above 1 mg/m^3). The following studies represent the types of responses across particle types and reflect the range of morphological responses reported.

Morphological changes were seen in the centriacinar regions of the lungs of cats exposed for 27 months to diesel exhaust (~6-12 mg/m^3), including the appearance of ciliated basal and Clara cells (Plopper et al., 1983). Following a six-month recovery period, peribronchiolar fibrosis in the proximal acinar region was reported in these same animals (Hyde et al., 1985). Guinea pigs exposed to diesel exhaust (~6 mg/m^3) for eight weeks showed hyperplasia of the alveolar epithelium and goblet cell hypertrophy (Wiester et al., 1980). An increase in type II cells and an accumulation of inflammatory cells in the alveoli and alveolar septal walls were seen in rats similarly exposed (White and Garg, 1981).

Kleinman et al. (1995) exposed rats to ammonium sulfate (20 or 70 μg/m^3, 0.2 μm MMAD), ammonium nitrate (90 or 350 μg/m^3, 0.6 μm MMAD), resuspended road dust (300 or 900 μg/m^3, 4 μm MMAD), or purified air, 4 h/d, 4 d/wk for 8 weeks. Lung permeability, as determined from measurements of albumin concentrations in bronchoalveolar lavage fluid, was significantly greater in rats exposed to high concentrations of road dust and nitrate, but not sulfate, when compared to air controls. Quantitative histopathologic analyses, which included measurements of alveolar nuclear density, alveolar chord length, alveolar septal thickness, and alveolar cross sectional area, showed moderate to substantial changes following exposure to particles. The severity of the responses was in the order of sulfate>nitrate>road dusts.

In a 24 month study of the exposure of rats and mice to diesel exhaust (0.35, 3.5 and 7 mg soot/m^3), a chronic inflammatory response was indicated by dose-dependent increases in inflammatory cells, cytoplasmic and lysosomal enzymes and lavageable protein at the two higher levels of exposure. Histologically, the rats developed focal areas of fibrosis associated with deposits of the soot, whereas the mice had only fine fibrillar thickening with occasional alveolar septal thickening in the high-level exposure group (Henderson et al., 1988). Similar lesions have been reported in other studies using similar exposure levels (Heinrich et al., 1986a, 1986c; Ishinishi et al., 1986; Mauderly et al., 1987; Brightwell et al., 1989; et al., 1993).

In rats exposed to aerosolized fluidized bed coal combustion fly ash (3.6 μm MMAD, 36 mg/m^3) for two or four weeks, morphological changes reported included thickening of the alveolar walls and perivascular inflammation (Shami et al., 1984). Additionally, small granulomas were observed in the alveolar region and the lung and bronchus-associated lymph nodes at 42 weeks post-exposure.

Exposure to more cytotoxic metal particles leads to more profound effects, although such effects may be related more to the inherent toxicity of the metal than to the "particle" effect. The nickel compounds provide an example of this toxic process. In rats and mice, 12 d exposures to aerosolized nickel subsulphide (~2-3 μm MMAD, 10 mg/m^3) caused a necrotizing pneumonia, occurring mainly in the centriacinar regions of the lung (Benson et al., 1987). In a subsequent study in rats and mice, histopathological changes in the lungs, including chronic active inflammation, fibrosis and alveolar macrophage hyperplasia, were associated with exposure to nickel oxide, subsulphide and sulphate hexahydrate.

The order of toxicity of the compounds studied corresponded to their water solubility (nickel sulphate > subsulphide > oxide) (Dunnick et al., 1989). Degeneration of the olfactory and respiratory epithelium, including the alveolar epithelium, was seen in rats exposed to cobalt sulphate (~1 μm MMAD, 0.3-30 mg/m^3) for 13 weeks (Bucher et al., 1990).

The effect of particle size and lung burden on the fibrotic response to silica was shown in a study in which mice were exposed to 0.74 μm particles over four weeks, resulting in a reversible inflammatory response; in contrast, 2 μm particles at the same mass burden (3.2 mg/m^3) induced no response. At a higher mass burden (10.7 mg/m^3), mice exposed to the 0.74 μm silica showed progressive inflammation with a significant perivascular influx of T-lymphocytes leading to focal fibrotic lesions (Velan et al., 1993). Exposure to titanium dioxide (0.28 μm MMAD, 3.9 mg/m^3) showed no effects.

In chronic studies with silica, Muhle et al. (1991) investigated the fibrogenicity of toner (4 μm MMAD, 1-16 mg/m^3), titanium dioxide (1.1 μm MMAD, 5 mg/m^3) and silicon dioxide (1.4 µm MMAD, 1 mg/m^3) in the lungs of rats exposed for 24 months. Using a dosimetric model, these investigators concluded that the relative fibrogenic potency of titanium dioxide, toner and silica was 1:5:418. No evidence of fibrosis was obtained with the animals exposed to the lowest toner concentration studied. A similar fibrotic response has been reported in rats exposed to aerosols of Ludox colloidal silica (2.9-3.7 µm MMAD, 10-150 mg/m^3) for four weeks (Lee and Kelly, 1992) and to cristobalite (1.4 µm MMAD, 27.5 mg/m^3) 50 weeks after an eight-day exposure (Absher et al., 1989).

Wolff et al. (1988) exposed rats to gallium oxide (Ga_2O_3) particles (0.2 µm, 23 mg/m^3) for four weeks and reported that at six months post-exposure, inhaled gallium oxide produced cytotoxic, inflammatory and fibrogenic responses similar to or greater than responses to inhaled quartz particles. The gallium oxide aerosols consisted of aggregates of ultrafine (14 nm) singlet particles.

Concerning soluble particle matter, of note is the minimal histological effects observed in rabbits exposed for one year (2 h/d, 5 d/wk) to sulphuric acid aerosols at a dose of 125 µg/m^3, (0.3 µm MMAD) (Schlesinger et al., 1992a), and 250 µg/m^3 (1 h/d, 5 d/wk)(Gearhart and Schlesinger 1989). The major responses produced by 125-250 µg/m^3 of H_2SO_4 are characterized by hyperplasia of epithelial secretory cells and hypertrophy in small airways (Gearhart and Schlesinger 1989; Schlesinger et al., 1992).

10.3.2 Biochemical Changes

Acute and Short-Term Exposure

The biochemical assays of lavage fluids of rats intratracheally instilled with silica (2.2 µm, 0.2-5 mg/100 g body weight), titanium dioxide (2.2 µm, 1 or 5 mg/100 g body weight) or aluminum oxide (5.3 mm, 1 or 5 mg/100 g body weight) indicated increased levels of lactate dehydrogenase, β-glucuronidase, N-acetylglucosaminidase and total protein, in a dose-dependent manner (Lindenschmidt et al., 1990). In an inhalation study using titanium dioxide (1.0 µm MMAD, 50 mg/m^3), no such effects were seen (Driscoll et al., 1991). The reason for the differing results is not clear. Elevated total protein is indicative of increased permeability of the alveolar-capillary barrier, whereas the elevated lactate dehydrogenase reflects cellular damage (Henderson et al., 1978). A concentration-dependent elevation in lactate dehydrogenase levels, resulting from *in vitro* treatment of alveolar macrophages with quartz (<5 µm), has also been reported, indicating that alveolar macrophage damage also contributes to increased lactate dehydrogenase levels (Chen et al., 1991). The increased lysosomal enzyme levels are believed to reflect elevated alveolar macrophage activity/numbers (Henderson et al., 1988). In most cases, these changes are seen as sensitive indicators of histological changes in the lung.

Similar increases in both total protein and enzyme levels were reported in rats after intratracheal instillation of 0.5-200 µg lanthium chloride (Suzuki et al., 1992), cobalt particles (4 µm, 0.06 mg/100 g body weight), tungsten carbide (2 µm, 1 mg/100 g body weight), tungsten carbide-cobalt powder (2 µm, 1 mg/100 g body weight) (Lasfargues et al., 1992) and a series of nickel compounds (0.01-1.0 µm Ni), including nickel subsulphide, nickel chloride, nickel sulphate and nickel oxide (Benson et al., 1986).

Other responses measured in bronchoalveolar lavage (BAL) fluid include elevated levels of phospholipid in rats following silica (6 mg) instillation, which was related to the surface-complexed iron (Fe^{3+}) (Ghio and Hatch, 1993); increased phospholipid in rats following inhalation of diesel exhaust (0.2 µm MMAD, ~6 mg/m^3) (Wright, 1986); and *in vitro* rat alveolar macrophage release of glutathione in response to quartz (Boehme et al., 1992).

Acidic aerosols, both fine (0.3 µm MMAD) and ultrafine (0.04 µm MMAD), were found to initially elevate lavageable protein, β-glucuronidase and TNF-α; however, the ultrafine aerosol was found to decrease *in vitro* alveolar macrophage phagocytosis, whereas the fine aerosol increased phagocytosis (Chen et al., 1992b).

Alveolar macrophages from rats exposed to silicon dioxide (0.9 µm MMAD, 45 mg/m^3) for eight days were found, five to seven months later, to have elevated levels of cyclo-oxygenase metabolites, including prostaglandin E_2, thromboxane B_{22} and prostaglandin D_2, "priming" the alveolar macrophages for TNF-α release (Mohr et al., 1992). Silica (1.6 µm MMAD) was also found to cause increased fibronectin and lipopolysaccharide-stimulated release of interleukin-1 (IL-1) (Driscoll and Maurer, 1991). In a summary of a number of such studies, Driscoll and Maurer (1991) suggest that IL-1, TNF-α and fibronectin correlate to exposures to toxic dust and may play a role in the pulmonary response. Increased TNF-α release has also been reported in hamsters exposed to opsonized albumin-coated latex particles (0.1 μm) (Kobzik et al., 1993).

Subchronic and Chronic Exposure

The types of biochemical changes reported in subchronic and chronic studies are similar to those seen in the shorter-term exposures and reflect similar processes. In guinea pigs exposed to silica (6-28 mg/m^3, 75% of particles below 2 μm) or titanium dioxide (24 mg/m^3, 85% of particles below 2 μm) for three weeks, the silica-exposed animals showed elevated enzyme levels in the BAL fluid eight weeks post-exposure. The titanium dioxide induced no effects (Sjöstrand and Rylander, 1984). Elevated levels of cytoplasmic and lysosomal enzymes and total protein in the BAL fluid were similarly reported in rats up to 12 months after exposure to 0.2 μm (aggregates of 14 nm particles, as above) gallium oxide particles (23 mg/m^3) over four weeks (Wolff et al., 1988).

The fibrosis reported by Henderson et al. (1988), discussed above under histopathological effects, was accompanied by elevations in lavageable β-glucuronidase and hydroxyproline. Lavageable glutathione and glutathione reductase activities increased in both species, but the extent of these effects was greater in mice. Whole-lung glutathione was depleted in a dose-dependent manner in rats, whereas it was increased in mice. Conceivably, this depletion may have played a role in the fibrotic response seen in the rat. Lung P-450 content was not elevated in either species.

Kuhn et al. (1990) exposed rats to bituminous coal dust (4-5 μm MMAD, 80% of particles between 2 and 7 μm, 25 mg/m^3) for two weeks. Exposure to the dust resulted in increases in lavageable alveolar macrophage numbers, which, *in vitro*, showed increased production of thromboxane A_2 and leukotriene B_4 and a decrease in prostaglandin E_2. The alteration in thromboxane A_2 and leukotriene B_4 production persisted for two weeks post-exposure.

10.4 PARTICLE-ASSOCIATED INCREASES IN MORTALITY

Acute exposures to some types of particles can result in increases in mortality. A number of the heavy metal particles are lethal, by virtue of their inherent toxicity – e.g., tri- or pentavalent arsenic (LD_{50} 14.3 mg/kg), vanadium pentoxide (LD_{50} 8.1 mg/kg) and selenium and cadmium oxides (LD_{50} 0.88 mg/kg) (Rhoads and Sanders, 1985). In terms of "particle effect" toxicity, increases in mouse mortality have been reported to occur after the intratracheal instillation of only 0.4 mg of 0.4 μm diesel soot (Sagai et al., 1993), although the chemical composition of the particles played a role in mediating this response, as methanol-washed diesel particles caused no increases in mortality.

For $(NH_4)_2SO_4$ and H_2SO_4, two important components of ambient particles, fairly high concentrations are required to induce animal acute mortality. Above 4 mg/m^3 of H_2SO_4 is required for acute lethality of guinea pigs, a species believed to be the most sensitive. The animal mortality induced by H_2SO_4 is primarily due to laryngeal or bronchoconstriction (Office of Air Quality Planning and Standards et al., 1982). Pure ammonium sulphate particles are rather inert, and exposure to $(NH_4)_2SO_4$ at 1000 – 2000 mg/m^3 for 3 days did not cause death in rats (Pepelko et al., 1980).

The most lethal particles investigated to date are those ultrafine particles that are freshly generated as fumes from some polymers and perhaps metals. Oberdörster et al. (1994a) exposed rats to Teflon® (26 nm MMAD, ~60 μg/m^3) for 10 minutes at inhaled number concentrations of 5 × 10^5 particles/cm^3, which resulted in acute mortality from a hemorrhagic pulmonary edema. Factors that appear to impact on the toxicity of these types of particles include particle aging and aggregation (Warheit et al., 1990; Lee and Seidel, 1991). The relevance of these observations to environmentally exposed humans remains open to speculation; however, ultrafine particles are consistently present in the urban atmosphere (Brand et al., 1992). Also, humans exposed to polymer fumes and metal fumes are known to develop symptoms observed in the animals (i.e., polymer fume fever and metal fume fever) (Shusterman, 1993; Gordon and Fine1993).

A more recent study using concentrated air particles shows that ambient particles concentrated and instantaneously given to rats for inhalation may cause death (Godleski et al., 1996). In this study, normal rats, rats with monocrotyline-induced pulmonary inflammation (50 mg/kg subcutaneously), and rats with SO_2-induced chronic bronchitis (250 ppm SO_2 for 6 weeks) were exposed to concentrated air particles (~250 μg/m^3) for 3 consecutive days, 6 h/d. Death occurred during exposure and overnight. The mortality for healthy rats, rats with pulmonary inflammation, and rats with chronic bronchitis were

0, 19% and 37%, respectively. Dead animals had a significantly more prominent airway constriction than did live animals. It is speculated that reactive oxygen species accompanying fresh ambient particles in concentrated form may play an important role in the acute mortality. Thus, ambient particle inhalation can cause death in rats with disease, and airway constriction appears to be important in the response.

Few investigators have noted an increase in mortality following subchronic or chronic exposures of animals to particles. Even in instances in which such exposures have resulted in the development of pulmonary fibrosis and lung tumours, increases in mortality have not been concurrently observed as a major outcome of exposure.

10.5 PARTICLE-INDUCED CARDIOVASCULAR RESPONSES

Since epidemiological studies conducted in Canada (Burnett et al., 1995) and in the US (Schwartz and Morris 1995) have shown a consistent association between fine particulate matter air pollution and increased hospital admissions due to cardiovascular diseases, several studies using experimental animals have been carried out in an attempt to investigate the underlying mechanism of ambient particle-induced cardiovascular effects.

Animal studies demonstrate that emission residual oil fly ash causes dose-related increases in electro cardiographic (ECG) abnormalities in rats (Campen et al., 1996) and dogs (Nearing et al. 1996). In the study conducted by Campen et al. (Campen et al., 1996), healthy rats and rats with monocrotaline-induced pulmonary hypertension (60 mg/kg) were instilled with residual oil fly ash (ROFA) intratracheally at doses of 0, 0.25, 1, or 2.5 mg. Data show that body core temperature and heart rate decreased immediately post-instillation in a ROFA dose-dependent manner. Analyses of ECG's show dose-related increases in arrhythmias and skipped heart beats. Pulmonary hypertension may contribute to the adverse health effects of particles.

Nearing et al. (1996) exposed healthy adult mixed-breed dogs to a fine particle aerosol (MMAD ~ 3 μm) produced by an aerosol generator using oil-fired power plant ash, for 3 consecutive days, 3 h/d. The particle concentrations were 1.68 or 3.04 mg/m^3. Measurements of potentially toxic metals (vanadium, nickel, zinc, copper and manganese) showed no acute increases with exposure. Dogs exposed to the higher concentration of particles had significant increases in the amplitude of T-wave altemans in ECG, an index of vulnerability to ventricular fibrillation, compared to pre-exposure control measurements. All dogs exposed to particles exhibited ST-segment elevation, while controls did not show any changes.

An *in vitro* study using isolated atrium of guinea pigs has shown that diesel exhaust particles (0.01 - 0.5 mg/ml) caused a negative inotropic action followed by the cardiac arrest of the isolated atrium (Sakakibara et al., 1994).

The mechanism for particle-induced cardiovascular response is not yet clear. However, diesel exhaust particles have been reported to cause oxidative modification of low density lipoprotein *in vitro* (Ikeda et al., 1995). Oxidized low density lipoprotein is known to be very cytotoxic (Morel et al., 1983; Ocana, 1989; Ikeda et al., 1995), inducing endothelial damage, the proliferation of smooth-muscle cells, causing monocyte-endothelial interactions (Berliner et al., 1990; Cushing et al., 1990), and platelet aggregation, which all eventually may lead to adverse cardiovascular effects. Indeed, intratracheal instillation of diesel exhaust particles (0.4 mg or 0.8 mg per mouse) caused acute disruption of capillary endothelial cells and the detachment from their basement membrane (Ichinose et al., 1995). An epidemiological study also shows an increased plasma viscosity during an air pollution episode (Weinmann et al., 1995). Oxidized low density lipoprotein is also demonstrated to be a potent inhibitor of endothelial-derived relaxation of vascular smooth muscles (Ezaki et al., 1994). Thus diesel exhaust particle-induced oxidization of low density lipoprotein may play a pathological role in promotion of coronary vasospasm and hypertension through inhibition in endothelium-dependent relaxation.

10.6 EFFECTS OF PARTICLES ON COMPROMISED LUNGS

Early studies conducted using animals with compromised lungs focussed on the changes to the deposition and clearance patterns of particles, and relied on artificially induced lung lesions as models for human disease conditions.

Ferin (1971) used papain to induce emphysema in rats and found that the deposition of aerosolized titanium dioxide (0.25 μm) was approximately one-half the amount deposited in normal lungs; the papain-treated lungs cleared ~18% of the deposited material as of 30 days after exposure, whereas normal lungs cleared ~51% of the originally deposited lung burden.

The deposition pattern of an inhaled submicrometre aerosol (^{99m}Tc-sulphur colloid, 0.46 µm MMAD) was studied in hamsters experiencing elastase-induced emphysema (Sweeney et al., 1987). Fewer particles were found deposited in the emphysematous lungs, and the pattern of deposition in the emphysematous lungs was less uniform than in control lungs. A similar effect was reported in hamsters with bleomycin-induced fibrosis, exposed to submicrometre-sized gallium oxide aerosol (Sweeny et al., 1983) or to submicrometre-sized particles of colloidal gold (Tryka et al., 1985). In the latter case, the particle clearance over five days post-exposure compared with day 0 exposure, was 45% in the fibrotic lungs, and 26% in the normal lung. The authors suggested accelerated alveolar-bronchiolar transport of particles or particle-containing macrophages may be responsible for the greater clearance in the diseased lungs.

In contrast, Greene et al. (1987) found that chronic bronchitis induced by a 60-week exposure (2 h/d, 5 d/wk) to 500 ppm of sulphur dioxide in dogs resulted in a lower deposition of inhaled ^{134}Cs-labelled aluminosilicate particles, yet their subsequent clearance from the lung was unaffected by the pre-existing disease. However, as only two sulphur dioxide-exposed and two unexposed dogs were involved in this study, the result is not very conclusive.

The influence of pre-existing emphysema on the susceptibility to the effects of chronic exposure to diesel exhaust was investigated by Mauderly et al. (1990). Rats with elastase-induced emphysema were exposed to diesel exhaust (3.5 mg soot/m^3) or air only for 24 months (7 h/d, 5 d/wk). The overall results of this detailed study did not support the hypothesis that emphysematous lungs are more susceptible than normal lungs to chronic exposure to high levels of diesel exhaust. It appeared that the decreased accumulation of particles in the emphysematous lungs may have negated any potential increase in response.

Little is known about how lung defence mechanisms against deposited particles may be altered in a background of acute lung inflammation. Slauson et al. (1989a) investigated the clearance of inhaled cobalt oxide particles (0.54-0.65 µm) using calves experiencing acute lung injury induced by parainfluenza-3 virus. Control animals had a biphasic clearance pattern, with rapid initial clearance of 50% of the initial lung burden as of seven days after exposure followed by a slower clearance component. With the virally infected calves, 90% of the initial lung burden was retained as of seven days after exposure. Virtually all of the particles lavaged from the control and experimental animals were contained in alveolar macrophages. However, histological analyses revealed that some particles were contained in macrophages in the interstitial regions of the lungs of calves with viral pneumonitis, and also increased transport to the regional lymph nodes; this outcome was not observed in the lungs of healthy calves.

In another study, Slauson et al. (1989b) studied the phagocytic activities of alveolar macrophages lavaged from the lungs of calves that were experiencing experimentally induced interstitial adjuvant pneumonitis. Compared with alveolar macrophages from healthy control animals, the alveolar macrophages from the inflamed lungs showed a higher maximum rate of particle (microbeads, 2.02 µm) phagocytosis *in vitro*, and a higher proportion of them were able to phagocytize multiple particles. These observations differ from those made by the same group in a study in which alveolar macrophages from calves experiencing virus-induced acute bronchiolitis and alveolitis were found to have depressed phagocytic activities.

Recently, Rabbe et al. (1994) exposed rats with elastase-induced emphysema to two particle atmospheres, a California-type aerosol [consisting of particles of graphitic carbon, natural clay, NH_4HSO_4, $(NH_4)_2SO_4$, NH_4NO_3, $PbSO_4$, $VOSO_4$, $MnSO_4$, and $NiSO_4$, MMAD 1.1-1.5 µm], and a London-type aerosol [consisting of NH_4HSO_4, $(NH_4)_2SO_4$, coal fly ash, and lamp black carbon, MMAD 0.8-0.9 µm]. While a 3 d exposure (23 h/d) to the London aerosol produced a significantly greater increase in pulmonary DNA and RNA contents in emphysema rats than in normal rats, there were no changes in tracheobronchial clearance or lung permeability compared to normals. A 30 d exposure to the California aerosol enhanced small airway lesions in emphysema rats, but did not alter lung hydroxyproline, tracheobronchial clearance, or small airway fibrosis.

More recently, Gilmour et al. (1997) examined whether residual oil fly ash (obtained from the stack of an oil fired power plant) could exacerbate the severity of influenza infection in rats. Female rats were given an intratracheal instillation of 1 mg residual oil fly ash along with an intranasal infection with influenza virus. Virus infection alone caused mild pulmonary inflammation and increased airway reactivity 24 hours post-infection that subsided by 3 days. Residual oil fly ash instillation resulted in a stronger inflammatory response and airway reactivity that persisted at the three day time point. The combination of residual oil fly ash with virus did not affect the clearance of virus from the lung, but resulted in significantly higher numbers of inflammatory cells at day 1 than either treatment alone and a heightened airway reactivity at three days. The data indicate that the dual insult of particulate matter and virus produces more severe respiratory symptoms and lung damage than either stimulus alone. However, the effects do not appear to be more than additive.

10.7 PARTICLE-INDUCED LUNG CANCER

10.7.1 Carcinogenicity Studies

The interpretation of experimental studies relating to the carcinogenic effects of particulate matter is confounded by the fact that the studies are necessarily conducted using a single particle type. In these long-term studies, the contribution to the overall effect due to the inherent chemical toxicity of the particle itself, rather than the more general "particle effect," is more difficult to assess. For example, a number of metals that are found as components of the particle mass have been classified as carcinogenic or potentially carcinogenic to humans by the inhalation route, including some of the inorganic compounds of arsenic (Health Canada, 1989), cadmium (CEPA, 1994a), chromium VI (CEPA, 1994b) and nickel (CEPA, 1994c). This review will focus only on carcinogenicity as it has been investigated in terms of a "particle effect" of inert particles. Diesel particles have been examined, as there is some evidence of a particle-related effect.

Another confounder is the carcinogenic potential of substances adsorbed to the particle surface – e.g., PAHs on diesel particles. Again, this review is focussed on the "particle effect" alone and will deal only briefly with this issue as it relates to diesel particles.

Pott et al. (1994) instilled 30-150 mg of diesel soot, carbon black, quartz, titanium dioxide and black iron oxide (Fe_3O_4) into rats' lungs with weekly instillations and found significant increases in lung tumour incidence in all exposed animals except for the titanium dioxide-instilled animals. Carbon black and diesel soot instillations resulted in similar lung tumour incidence, indicating that the diesel exhaust-induced lung tumours seen in rat inhalation studies may be explained by a particle effect. Kawabata et al. (1986) also observed that both diesel soot and carbon black can induce lung tumours in rats after intratracheal instillation.

Heinrich et al. (1986a, 1986b) performed a two-year chronic inhalation study in female mice and rats and in both sexes of hamsters with filtered and unfiltered diesel exhaust (4.0 mg/m^3, 19 h/d, 5 d/wk). No tumours were found in the lungs of hamsters. A significantly increased incidence of malignant tumours over the control group was observed in the lungs of both diesel exhaust-exposed mice (adenocarcinoma) and rats (squamous cell tumour). In satellite groups of animals additionally treated with benzo[a]pyrene (BaP), dibenz[ah]anthracene or diphenylnitrosamine, syncarcinogenic effects of diesel exhaust were found only in the respiratory tracts of rats.

A number of investigators in Europe, the United States and Japan have confirmed that inhalation of diesel exhaust at concentrations exceeding ~2 mg/m^3 for two years can induce lung tumours in rats. The study design was essentially similar, in that freshly generated diesel engine exhaust was diluted and rats were exposed in whole-body inhalation chambers to differing concentrations ranging from 0.3 to 7 mg/m^3. In some studies, mice and hamsters were also exposed under similar conditions. Collectively, these studies showed that rats, but not mice and hamsters, had significantly increased lung tumour rates when they were exposed to the unfiltered diesel exhaust (Ishinishi et al., 1986; Heinrich et al., 1986b; Mauderly et al., 1987; Brightwell et al., 1989; Kawabata et al., 1993).

Chen and Vostal (1984) exposed rats to diesel particles (1.5 mg/m^3 and 6 mg/m^3) for 2-28 days (20 h/d, 7 d/wk) to investigate aryl hydrocarbon hydroxylase (AHH) activity in their alveolar macrophages. Additionally, AHH activity in alveolar macrophages after the intratracheal instillation of BaP (5 mg/kg body weight) or diesel exhaust extract was also investigated. After four weeks of exposure to both diesel exhaust concentrations, AHH activity in alveolar macrophages was decreased. BaP instillation resulted in a slight increase in AHH activity in alveolar macrophages, whereas the diesel extract resulted in no change in AHH activity. The results of this study suggest that the amounts of aromatic hydrocarbons associated with the diesel particles were insufficient to induce AHH activity in the alveolar macrophages.

The question as to whether the results of the diesel exhaust studies in rats are attributable to the particle phase only or whether the organic compounds adsorbed on the particles also play a role was addressed in studies by Heinrich et al. (1992). Female rats and mice were exposed for two years (18 h/d, 5 d/wk) either to diesel soot or carbon black at concentrations ranging from 0.8 to 7.5 mg/m^3 or to ultrafine titanium dioxide particles (20 nm) at average concentrations of 12 mg/m^3. The investigators found that only rats developed both benign and malignant tumours after exposure to all three particle compounds (diesel, 22%; carbon black, 39%; titanium dioxide, 32% combined tumour incidence in high-dose groups), which indicates that the particle compounds, including the ultrafine titanium dioxide, have a carcinogenic potential in rats and that the diesel exhaust-induced lung tumours can be fully explained by the particles.

Lee et al. (1985a) exposed rats to titanium dioxide (1.5-1.7 µm MMAD at 0, 10, 50 and 250 mg/m^3) for two years (6 h/d, 5 d/wk). Bronchoalveolar adenomas and cystic keratinizing squamous cell carcinoma occurred in ~17% of the rats exposed to the 250 mg/m^3 concentration of titanium dioxide, with no increases seen at lower dose levels.

In contrast to the study by Heinrich et al. (1992) with ultrafine titanium dioxide, the titanium dioxide particles used by Lee et al. (1985a) were more than a factor of 10 larger. Lung tumours induced by high particle burdens of benign particles such as titanium dioxide can be associated with a lung particle overload effect (Morrow and Mermelstein, 1988; Morrow, 1994). However, even benign particles can become significantly more toxic if their size is in the ultrafine particle range, and, as demonstrated by the Heinrich et al. (1992) study, 20-fold lower inhaled concentrations in the rat than used in the particle overload study by Lee et al. (1985a) will induce lung tumours.

Muhle et al. (1989) exposed rats for two years (6 h/d, 5 d/wk) to crystalline silica (1 mg/m^3), test toner (0, 1, 4 and 16 mg/m^3) or titanium dioxide (5 mg/m^3). They observed that 18% of the silica-exposed rats developed benign and malignant lung tumours, significantly increased above the tumour rate of controls. No increases in tumour incidence were reported for the test toner or the titanium dioxide.

Reuzel et al. (1994) reported that chronic exposure of rats to aerosolized polymeric methylene diphenyl diisocyanate (<5 µm, 0, 0.2, 1.0 or 6.0 mg/m^3) for up to 24 months (6 h/d, 5 d/wk) caused lung tumours in the high-dose group (6 mg/m^3).

The overall evidence indicates that a number of different particle types, including titanium dioxide, carbon black, diesel soot particles and silica, when inhaled at sufficient dose levels, can induce lung cancer in rats, but not in mice or hamsters. The studies conducted with diesel particles show that the particle itself, rather than the chemical constituents or adsorbed chemicals, may be important in the carcinogenic process. Given the very high particle loads required to induce a carcinogenic response, it is unclear how these studies could be interpreted with respect to their potential extrapolation to humans.

10.7.2 Mutagenicity

Although genotoxic effects of particles have been associated with specific carcinogenic metal compounds, as discussed above, these would not be considered strictly particle effects. Potential particle-related genotoxic effects have been reported in studies examining diesel exhaust. Bond et al. (1990) examined rat alveolar type II cells from animals exposed to diesel particles or carbon black at 6.2 mg/m^3 for 12 weeks (16 h/d, 5 d/wk) and found a four-fold increase in DNA adducts for both exposures. In an earlier study, Wolff and co-workers (1989) measured DNA adducts in the lungs of rats after they were exposed for 12 weeks (4 h/d, 5 d/wk) to pure aerosols of BaP (2 mg/m^3) or BaP adsorbed on carbon black particles (97 mg/m^3). Although there was no significant difference in levels of DNA adducts, some evidence was obtained to indicate that the pattern of the DNA adducts formed in response to BaP alone and BaP adsorbed onto the carbon particles was different.

Interaction of urban particulate matter with cellular macromolecules may occur, leading to DNA damage or enzyme induction. The formation of DNA adducts has been observed following incubation of urban particles from Ottawa, Ontario, 10 µg/ml medium, with normal human bronchial epithelial cells for 72 hours (Shah et al., 1995).

Vincent et al. (1995) incubated urban particles from Ottawa, Washington, D.C., or St. Louis, or aqueous extracts of $PM_{2.5}$ (100 µg/ml medium) *in vitro* with a panel of HepG2 cell lines transfected with target gene constructs, for 18-24 hours. Urban particles caused gene expression of several enzymes including cytochrome P4501A1, glutathione S-transferase Ya, and a xenobiotic response element, which are biologically critical in chemical biotransformation. The potency of gene induction was correlated with the content of polycyclic aromatic hydrocarbons in particles. The induction of gene expression of cytochrome P4501A1 and the xenobiotic response element was significantly potentiated by co-incubation with rat alveolar macrophages (Goegan and Vincent, 1995), indicating that alveolar macrophages may increase the bioavailability of polycyclic aromatic hydrocarbons from particles.

10.8 COMBINED EXPOSURES TO PARTICLES AND OTHER AIR POLLUTANTS

Most pulmonary toxicological studies of particles deposited as aerosols or by the intratracheal instillation route have involved exposures to single materials. However, "real-world" exposures to ambient environments involve combinations of a number of toxic and potentially toxic materials that may be inhaled together or more or less sequentially under some conditions. Possible interactions that may result from the inhalation of mixtures of toxicants conceivably can include simply additive effects, synergistic effects or even antagonistic effects. This section summarizes several studies in which combined exposures to test materials were assessed for their overall toxic effects as well as some studies in which exposure to one material was followed by exposure to another.

10.8.1 Combined Exposure with Ozone

Particulate matter and ozone are two major pollutants in ambient air. Several studies have examined how the injurious responses to sulphuric acid are altered by other pollutants. Warren et al. (1986) investigated the interaction between inhalation of ozone and an acid aerosol composed of ammonium sulphate. Rats were continuously exposed to ozone (0.2 ppm) with and without concurrent exposure to 5 mg/m^3 ammonium sulphate (0.5 μm MMAD) for two to seven days. A synergistic interaction between ozone and the acid aerosol was identified by greater than expected increases in the rate of hydroxyproline synthesis by lung tissue and lavageable protein. In another series of studies reported by Warren and Last (1987), a synergistic interaction was also observed between 0.2 ppm ozone and 100, 500 or 1000 μg/m^3 sulphuric acid aerosol after three days' exposure of rats, by the criterion of a significant increase in total lavageable lung protein. Synergistic interaction was also observed, by the criterion of increased lung protein content, in rats exposed to 0.2 ppm ozone plus 40, 100 or 500 μg/m^3 sulphuric acid aerosol for seven or nine days. Synergistic elevations in lung collagen were also observed when rats were exposed to 0.64 ppm ozone and 200, 500 or 1000 μg/m^3 sulphuric acid aerosol. The interaction between ozone and sulphuric acid evidently is one in which the damaging effects of the ozone in the centriacinar region of the lung are enhanced. In this case, the acid aerosol potentiates the effects of ozone and not vice versa.

In another study, Last et al. (1984) examined biochemical and morphometric changes in the lungs of rats exposed for three, seven or 14 days to ozone (0.64-0.96 ppm), with or without accompanying exposure to aerosolized ammonium sulphate (~1 μm MMAD, 5 mg/m^3). After three days of exposure to the mixtures, lung macrophages and monocytes increased two- to three-fold, fibroblasts increased two-fold and collagen synthesis rates increased 2.5-fold compared with values obtained from animals exposed to ozone only. Only relatively minor, if any, changes were observed with ammonium sulphate alone.

Schlesinger and co-workers (1992b) found that interactions between exposures to both sulphuric acid aerosol and ozone can be both antagonistic and synergistic. In their study, rabbits were exposed for three hours to submicrometre sulphuric acid aerosol (50-125 μg/m^3) with 0.1, 0.3 or 0.6 ppm ozone. An antagonistic relationship was demonstrated by depressed phagocytic activity of lavaged alveolar macrophages at the two highest concentrations of acid aerosol, whereas ozone alone did not affect this alveolar macrophage function. Exposure to 75 or 125 μg/m^3 sulphuric acid caused a reduction in tumor necrosis factor-α (TNF-α) production by stimulated alveolar macrophages, a response that was not observed with ozone only. All mixed exposures containing 75 μg/m^3 sulphuric acid also reduced TNF-α production by the alveolar macrophages, whereas exposures to mixtures of 125 μg/m^3 acid with 0.3 or 0.6 ppm ozone resulted in an enhanced level of production of this proinflammatory cytokine.

Chen et al. (1991a) investigated possible interactions between aerosolized sulphur oxides and ozone. In the first exposure regimen, guinea pigs were exposed for one hour to pure sulphuric acid (0.08 µm MMAD, 300 µg/m^3), to sulphuric acid layered on zinc oxide particles (0.05 µm CMD, 24 or 84 µg/m^3) or to zinc oxide particles only (6 mg/m^3); two hours later, they were exposed for one hour to air or 0.15 ppm ozone. The guinea pigs exposed to the 84 µg/m^3 concentration of acid layered on zinc oxide and subsequently exposed to 0.15 ppm ozone showed a more than additive reduction in vital capacity and diffusion capacity. In a second set of exposure regimens, guinea pigs were exposed to 24 µg/m^3 sulphuric acid layered on the zinc particles for five days (3 h/d). On days 8 and 9, the animals received two additional, three-hour daily exposures to the acid layer particles or a single one-hour exposure on day 9 to 0.15 ppm ozone. The second set of acid exposures induced greater decreases in lung volume and diffusion capacity than reported after the initial exposures. The ozone exposure induced reductions in lung volumes and diffusing capacity that were not observed in animals that received exposures to either sulphuric acid-layered particles or ozone alone. Overall, these studies indicate that single or multiple exposures to sulphuric acid-layered zinc oxide can sensitize guinea pigs to subsequent sulphuric acid or ozone exposures.

The effects of ozone exposure (0.8 ppm, 37 exposures, 6 h/d) on the fibrotic response elicited by intratracheally instilled silica (Min-U-Sil-5, ~1.5 µm, 2-50 mg) were examined in the rat by Shiotsuka et al. (1986). There was no detection of an interaction between silica and ozone in the development of pulmonary fibrosis, as determined by quantitative biochemical indices (hydroxyproline and lysyl oxidase) or by histopathological criteria.

Repeated exposure of rabbits to the mixture of H_2SO_4 (75-125 µg/m^3) and ozone (0.1-0.6 ppm), 3 hours per day for 5 days, induced synergistic/additive interactions in stimulating bronchial hyperresponsiveness and down-regulation of macrophage function (McGovern et al., 1993). In addition, repeated inhalation of mixture of ozone (0.4 ppm), H_2SO_4 (500 µg/m^3) and carbon particles (250 µg/m^3) for 5 days lead to greater lung parenchyma lesion and change in breathing pattern in rats compared with exposure to ozone alone, while there was no synergistic effect on depression of phagocytic activities of macrophages (Kleinman et al., 1993). Long-term exposure (Schlesinger et al., 1992) of rabbits to the mixture of H_2SO_4 (125 µg/m^3) and ozone (0.1 ppm) for 2 h/d, 5 d/wk for up to one year did not show any indications of interaction in terms of clearance response between acid and ozone in the group exposed to the mixture. Histological examination of intrapulmonary conducting airways showed an increase in secretory cell number after exposure to ozone alone or the mixture by 4 months, and there was evidence for synergistic interaction between ozone and acid. At subsequent times the interaction became antagonistic in nature with respect to histological changes (Schlesinger et al., 1992).

Bouthillier et al. (1996) exposed rats to a combination of ozone (0.8 ppm) and Ottawa particles (50 mg/m^3, MMAD 1.4 µm, collected from air conditioner filters) via nose-only inhalation for 1 or 3 days. Exposure to particles alone did not increase levels of protein, fibronectin and alkaline phosphatase in bronchoalveolar lavage fluid, nor did it exacerbate these effects of ozone following co-exposure. While exposure to ozone or particles alone altered some aspects of alveolar macrophage physiology, these cellular effects appeared to be attenuated by co-exposure of two pollutants. Particles potentiated the ozone-induced neutrophilic infiltration in lung lavage.

10.8.2 Combined Exposure with Other Pollutants

Castranova et al. (1985) exposed groups of rats to filtered air, coal dust (8.5 µm MMAD, 2 mg/m^3), diesel exhaust (~0.3 µm, 2 mg/m^3) and diesel exhaust (1 mg/m^3) plus coal dust (1 mg/m^3) for two years (7 h/d, 5 d/wk). Exposure to the coal dust resulted in an increase in lavaged alveolar macrophage numbers, increased the abilities of the alveolar macrophages to produce reactive oxygen and increased alveolar macrophage surface ruffling, which the investigators interpreted as indicating enhanced activity. The diesel exposure, on the other hand, decreased both the ability to generate reactive oxygen and cell surface ruffling. The combination of the coal dust with the diesel exposure resulted in a reactive oxygen generation potential that was essentially an average level between what was observed with the individual exposures.

The cytotoxic effects of carbon particles, sulphuric acid mist and the combination of the two on the tracheal epithelium were assessed by Schiff et al. (1979) using both *in vivo* and *in vitro* models. In the *in vivo* component, hamsters were exposed for three hours to an aerosol of carbon black (99.5% pure, 0.3 μm, 1.5 ± 0.4 mg/m^3) or sulphuric acid aerosol (0.12 μm MMAD, 1.1 mg/m^3), or both pollutants. Their tracheas were removed immediately, or at 24, 48 or 72 hours after exposure; or the animals were killed less than 1 hour after exposure and their tracheal rings were studied over a 72 hour period. In the *in vitro* component, ciliary beat frequencies of tracheal rings from normal hamsters were measured in response to exposure to carbon particles and sulphuric acid solution, alone or in combination. The ciliary beat frequency of the acid-carbon group in both components of the study was significantly reduced from the control and acid and carbon alone treatments. Histological analyses of the *in vivo* and *in vitro* samples showed that epithelial damage caused by acid mist-carbon exposure, including occasional rounded, swollen epithelial cells, focal losses of ciliated cells and elevations in mucosubstances, did not differ from that produced by acid mist alone, but the damage was greater than that produced by carbon only.

Alarie et al. (1973) exposed guinea pigs (whole body) and cynomolgus monkeys (face mask) to mixtures of fly ash (5 mg/m^3) and sulphur dioxide (0.1-5 ppm) for 52 weeks (23 h/d). This same group previously found that exposures to these materials alone do not cause deleterious effects. Likewise, no evidence was found that combined exposures to sulphur dioxide and fly ash significantly affected the pulmonary function status of the animals or other endpoints, including serum biochemistry and hematology and lung histopathology.

Inhaled fine-mode volcanic ash (0.65 μm MMAD, 9.4 mg/m^3), sulphur dioxide (2.7 mg/m^3) and a combination of sulphur dioxide and ash inhalation (2 h/d for 5 days) yielded no observable effects in normal rats and rats with elastase-induced emphysema (Raub et al., 1985). In a second part of the study, coarse (12.1 μm MMAD, 50 mg and 300 μg/rat) and fine (2.2 μm MMAD, 50 mg and 300 μg/rat) volcanic ash, pure silica (<5 μm, 50 mg and 100 μg/rat) and saline were intratracheally instilled into elastase-induced emphysematous and normal rats (Raub et al., 1985). Low- and high-dose injection of ash did not produce any indication of fibrosis. However, lung wet and dry weights increased and pulmonary alveolitis was observed after the high-dose instillations. In addition, silica-instilled rats showed changes consistent with interstitial lung fibrosis.

Murthy and Holovack (1991) investigated the ultrastructural effects of cadmium oxide (0.1 mg), alone and in combination with zinc oxide (5 mg), copper oxide (0.2 mg) and nickel oxide (1 mg), one week after these compounds were intratracheally instilled into the lungs of rats. Treatments with these heavy metals caused increases in alveolar macrophages and type II cell hyperplasia, but the general design of the study precludes any clear conclusion that the metals in combination are additive, synergistic or inhibitory.

Fujimaki et al. (1992) examined how exposure to sulphuric acid aerosol in combination with nitrogen dioxide might affect the functional properties of lung mast cells. They exposed guinea pigs to 1.0 mg/m^3 sulphuric acid and 4 ppm nitrogen dioxide for two weeks. Lung mast cell suspensions were then isolated, and antigen or A23187-induced histamine release was measured. No evidence was found that the combined exposure caused an increase in histamine release by the mast cells.

Last and Warren (1987) examined interactions between respirable aerosols of sulphuric acid (0.35 μm MMAD) or sodium chloride (0.38 μm MMAD) and nitrogen dioxide. They exposed (whole body) rats for one, three or seven days (23.5 h/d) to 5 ppm nitrogen dioxide, alone or in combination with ~1 mg/m^3 sulphuric acid or sodium chloride aerosols. A synergistic interaction was found between the nitrogen dioxide and sulphuric acid or sodium chloride exposure, as indicated by lung collagen synthesis (165% increase). Neither exposure alone produced such a response. A synergistic interaction was also shown by the criterion of increased protein content in lung lavage fluids from rats exposed to the nitrogen dioxide and 1 mg/m^3 sulphuric acid after one day of exposure and between the nitrogen dioxide and sodium chloride aerosol after three days of combined exposure.

10.9 ANIMAL TO HUMAN EXTRAPOLATION

The effects reported in experimental animal studies provide a basic understanding of the potential toxicological impacts of inhaled particulate matter. However, to use this information, both to predict potential effects in humans as well as to explain the results reported in human epidemiological studies, understanding those factors that affect the extrapolation of such effects to humans is important. Examining the differences between the experimental animal species themselves is also important, as this can impact upon how the effects are extrapolated to humans.

10.9.1 Differences in Respiratory Anatomy

In considering experimental animal studies, it is important to recognize that there are significant anatomical differences between the experimental animals and humans at all levels of the airway. Three anatomical regions are important when considering the response to the inhalation of particles: the extrathoracic, nose/mouth to the entrance of trachea; the tracheobronchial, trachea to the terminal bronchioles; and the pulmonary, the parenchymal airspace of the lung, including the alveoli. The focus of the studies reported here is limited to those describing effects in the lower respiratory tract (tracheobronchial airway to parenchyma), as it is this region of the airway identified in the epidemiological studies as the target site. A detailed discussion of the inter-species differences in these regions is beyond the scope of this assessment; however, comprehensive review articles are available in the literature (Schlesinger and McFaddden, 1981; Phalen and Oldham, 1983; Tyler et al., 1985; Patra et al., 1986; Henderson et al., 1987; Plopper et al., 1989; Stone et al., 1992).

The fundamental difference in the lower respiratory tract between humans and experimental animals is that the branching of the airways in humans is primarily a regular dichotomous pattern, whereas it is monopodial in mice, hamsters, rats, dogs, horses, rabbits and monkeys. The dichotomous pattern consists of regular subdividing of the major airway into increasingly smaller airways, as opposed to the monopodial pattern, which consists of a long tapered airway with smaller side branches. These differences are important, as particles tend to accumulate at the branch points in the airway owing to impaction.

At the level of the peripheral airways, humans have several generations of well-defined bronchioles, whereas the bronchioles are ill-defined or not present at all in rats, mice and hamsters. As the bronchioles are the last generation of the conducting airway bearing cilia, this difference is manifest in differences in clearance kinetics (see below).

In addition, when assessing toxic responses to inhaled materials in rodents, it has to be kept in mind that rodents are obligatory nose breathers, whereas a significant fraction of humans are routinely mouth breathers or oro-nasal breathers (Niinimaa et al., 1981), and inspired airflow partitioning from the nasal route to the oral pathway in otherwise predominantly nose breathers occurs during many human activities (e.g., Saibene et al., 1979; Camner and Bakke, 1980; Niinimaa et al., 1981). Accordingly, results emanating from such studies may not realistically depict the responses that may occur in humans when defences offered by the upper respiratory tract are bypassed via mouth breathing during exposure to toxic gases or particles.

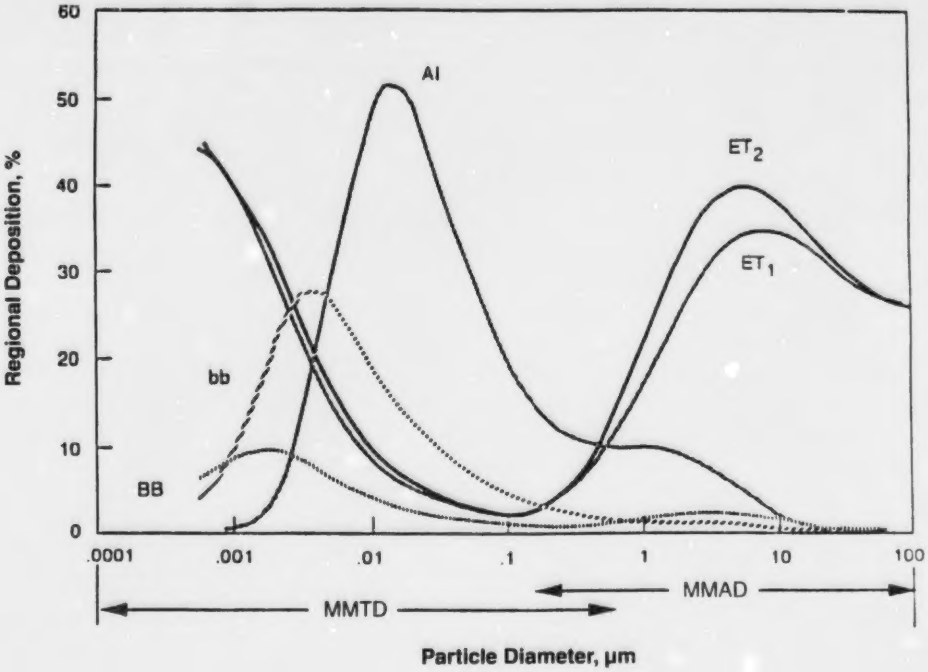
10.9.2 Deposition, Clearance and Fate

The deposition, clearance and fate of inhaled particles in the human airway have been extensively studied using low doses of a wide variety of relatively inert particles. Studies in experimental animals have tended to use very high doses to examine the effects of dose on the mechanisms of the deposition of the particles. A considerable effort has been made to model the deposition and clearance of particles within the respiratory tract.

Deposition

Deposition studies examine the rate and extent to which particles are delivered at the site of toxic action. Detailed reviews of the deposition of inhaled particles have been published by the US EPA (1982a) and more recently by Dodgson and McCallum (1994) and ICRP (1994). When considering the potential health impacts resulting from the inhalation of particulate matter, the regional deposition must be considered rather than just total deposition, owing to the very different clearance times within the regions and the different potential target tissues. The experimental data and modelling efforts are very well developed for deposition of airborne particulate matter, and the information base for humans has been summarized in Figure 10.1.

Figure 10.1 Deposition of inhaled particles in the human respiratory tract during nose-breathing, showing the high alveolar deposition for ultrafine particles of ~20 nm diameter in the AI region of the lung.



AI= alveolar-interstitial (including respiratory bronchioles, alveolar ducts and alveolar sacs); BB= bronche; bb=bronchioli; ET_1=anterior nasal; ET_2=posterior nasal; pharynx and larynx; MMAD=mass median aerodynamic diameter; MMTD=mass median thermodynamic diameter (adapted from ICRP,1994)

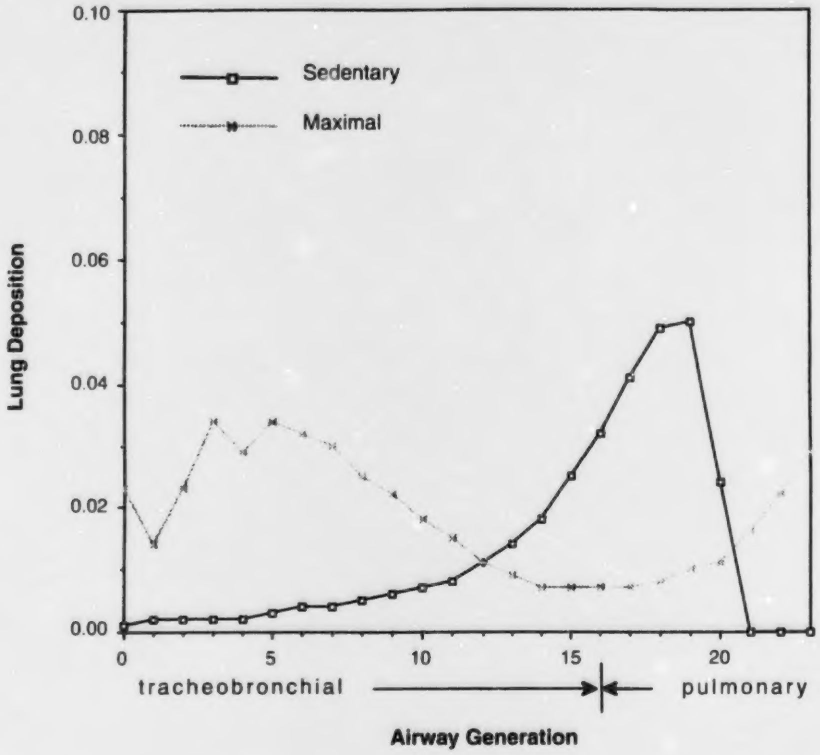
In the extrathoracic region in humans, virtually all particles >10 µm in diameter, when inhaled through the nose, are deposited in the nasal region, whereas during mouth breathing this drops to approximately 65% (US EPA, 1982a). Owing to the bypass of the nasal cavity during oral breathing, up to 10% deposition of particles up to 15 µm in diameter can occur in the tracheobronchial region (Miller et al., 1979). As impaction is the primary mechanism of deposition in the extrathoracic region, the deposition curve is highly dependent on flow rates.

In the tracheobronchial region, only particles <10 µm in diameter (bb and BB in Figure 10.1) are deposited; however, as noted above, during mouth breathing, particles up to 15 µm in diameter can deposit in this region.

The deposition of particles in the pulmonary region in humans is the most critical regarding the health effects associated with particulate matter. These generalized deposition fractions have also been shown to change as a function of breathing patterns. In a series of studies at a constant flow rate of 250 ml/s, increasing the residency time between two and eight seconds resulted in a decrease in the particle size showing the greatest probability of depositing in the lung as well as a decrease in the total deposition fraction (Heyder et al., 1980, 1986). It was also shown that the particle size of maximal deposition for the peak at approximately 3.5 µm diameter, as well as the absolute deposition fraction at the peak, decrease as flow rate increases (Heyder et al., 1980, 1986).

An example of the effect of changes in respiratory mechanics (through exercise) on the overall regional deposition of particles throughout the airway can be seen in Figure 10.2, where the trachea = generation 1; terminal bronchioles = generation 16; and alveoli = generation 23 (Martonen et al., 1992). The model predicts a significant shift in maximum deposition sites from the alveolar region of the respiratory tract towards the conducting airways when switching from sedentary to maximal physical activity in a human inhaling 2 µm-sized particles. The deposition patterns

Figure 10. 2 Model prediction of change in deposition of inhaled 2 μm diameter particles in human airways when physical activity is increased (Adapted from Martonen et al., 1992)



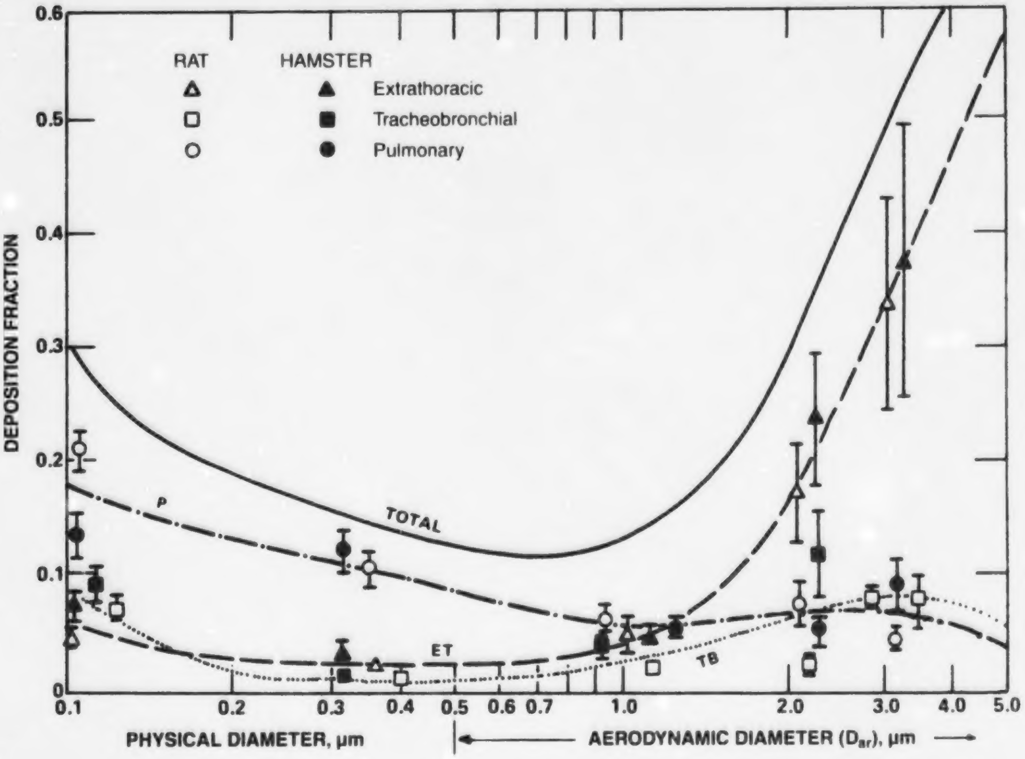
for intermediate levels of exercise are similar, with the pulmonary deposition occurring more deeply in the lung and an increasing amount of deposition in the tracheobronchial regions.

Modelling simulations have predicted significant age-dependent changes in the relative deposition of particles within the airway. Calculations based on diesel exhaust particles (0.2 μm) showed increased deposition in the nasopharyngeal region for children, compared with adults; maximal deposition was in the respiratory bronchiole region (airway 17) for newborns, whereas it was at the alveolar region (airway 20) for adults and children two years of age and older (Yu and Xu, 1987). Becquemin et al. (1987) measured total deposition of 1 or 2 μm polystyrene particles in children (<8 years and >8 and <15 years) and adults. The children, particularly those <8 years of age, showed higher deposition for spontaneous and quiet controlled breathing but showed lower deposition during exercise, with higher deposition in all cases for 2 μm versus 1 μm particles. Specific regional deposition was not examined.

Deposition in experimental animals is important both for understanding the toxicity of particles in animals and for extrapolating such studies to humans. Figure 10.3 summarizes the regional deposition data from Raabe et al. (1977) for rats and hamsters exposed to fused alumino-silicate spheres. It is important to note that particles >5 μm in diameter do not reach the tracheobronchial region and therefore would not be considered "inhalable" for rats or hamsters.

The effect of exercise on particle deposition in animals has also been studied. Exercising and resting rats were exposed for 30 minutes to ultrafine $^{67}Ga_2O_3$ particle aggregates (0.1 μm, 1.7 mg/m^3). Exercising rats had a lower lung deposition efficiency; however, as these rats also had higher inhaled volumes over the exposure period, resulting in more particles being inhaled, the final particle lung burdens in exercising and resting animals were not different (Hesseltine et al., 1986). Increased lung burden was demonstrated when nighttime and daytime particle depositions of gallium oxide aerosol were compared in rats. Greater deposition was found to occur during

Figure 10.3 Deposition of inhaled monodispersed aerosols of fused aluminosilicate spheres in small rodents showing the deposition in the extrathoracic (ET) region, the tracheobronchial (TB) region, the pulmonary (P) region, and the total respiratory tract based upon Raabe et al., (1977). (From EPA 1982)



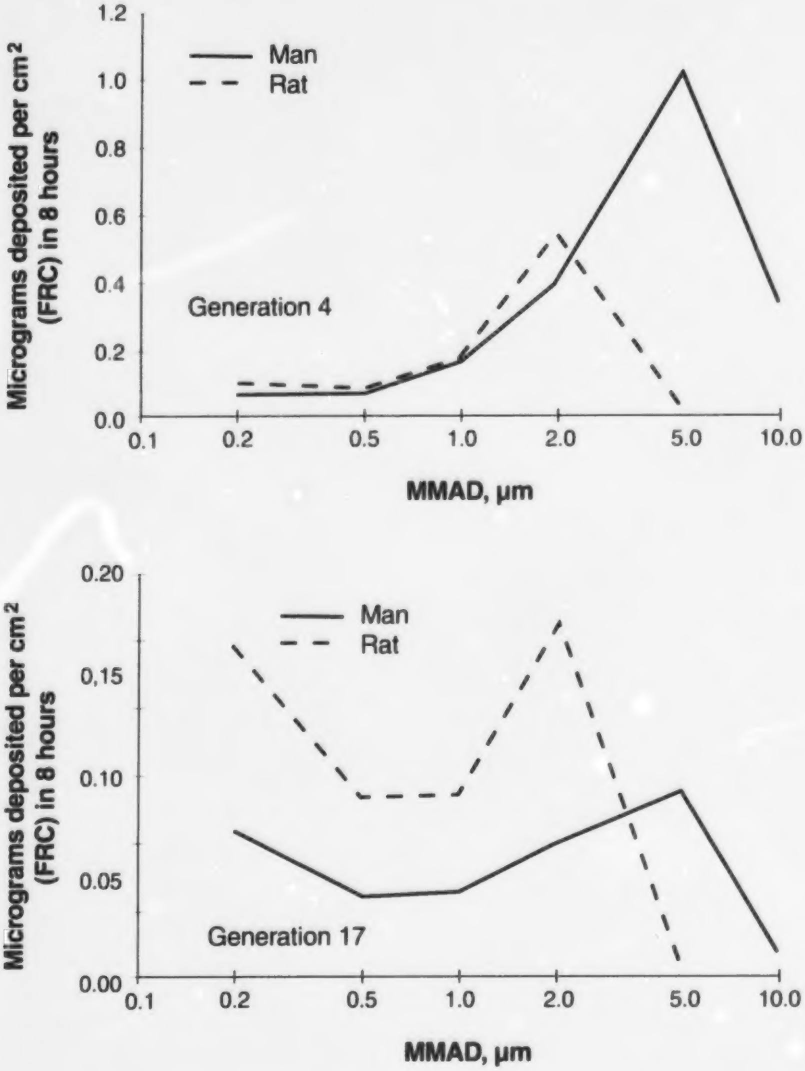
the nighttime exposures, which the investigators postulated may reflect an increased ventilation during the nocturnal activity common to rodents (Hesseltine et al., 1985). In exercising and resting hamsters exposed for 45 minutes to latex particles (0.9 μm, 4959 particles/cm^3), the exercising hamsters had significantly higher particle deposition in their noses, whereas the deposition in the alveolar region was unaffected by exercise compared with resting hamsters (Zeltner et al., 1991).

The fractional regional deposition efficiency alone does not necessarily reflect the actual dose received by a given region in the respiratory tract. Instead, the actual amount (dose) deposited per unit surface area of a given airway generation may be of much greater relevance for respiratory tract dosimetry. Figure 10.4 shows two examples of a deposited surface area dose in individual generations of the lower respiratory tract of rats and humans (Oberdörster et al., 1994b). Whereas for larger particles a greater surface area dose in the upper generations is achieved in humans, the opposite is true for the smaller particles (<~3 μm) depositing in the transitional zone of the lung (generation 17) (i.e., a greater surface area dose is reached here in the rat). These models predict significant differences in the deposited dose between specific airway generations in rats and humans. This difference can have significant implications for observed effects and further complicates the extrapolation of animal studies to humans.

Clearance and Fate

The mechanisms by which particles are cleared from each of the three regions of the airway discussed above are quite different. In the extrathoracic region, clearance occurs by mechanical processes; in the nasal area, by blowing, wiping or sneezing; and in the more anterior regions, either through swallowing (in mucus) or by expectorating. Clearance from the extrathoracic region may take up to one or more days in humans (Proctor and Wagner, 1965).

Figure 10.4 Model prediction of mass deposition rate per unit airway surface in generation 4 and generation 17 of the lower respiratory tract in rats and humans, as a function of inhaled particle size. Significant differences are apparent (inhaled concentration: 1 mg/m^3; nasal breathing at rest) (Oberdörster et al., 1994b).



In the conducting airways of the tracheobronchial region, the most prominent mechanism for elimination of particles is via the action of the mucociliary escalator into the gastrointestinal tract. This is a very fast clearance pathway, which is mainly completed within 24 hours after deposition of particles in this region. The half-time of clearance in humans ranges from half an hour in the larger airways to five hours in the smaller airways (US EPA, 1982a). However, there is some evidence of a long-term component to tracheobronchial retention (Stahlhofen et al., 1986a, 1986b; Smaldone et al., 1988); potential mechanisms for this long-term phase could be phagocytosis and subsequent retention by airway macrophages in the conducting airways or retention of particles in the sol phase of the mucus layer on top of the epithelial cells (Gehr et al., 1990; Lehnert et al., 1992).

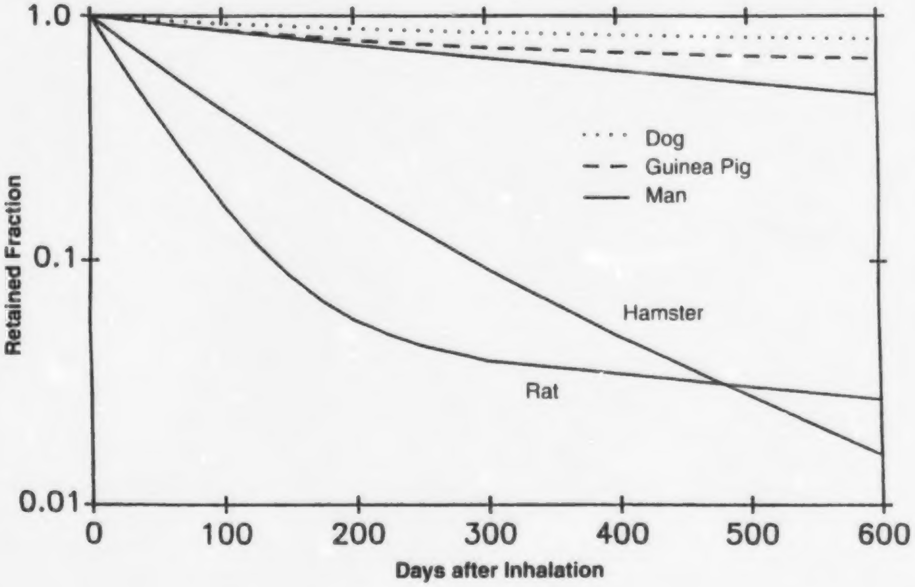
In the pulmonary region, insoluble particles are rapidly phagocytized by alveolar macrophages, or they may also enter the interstitium via endocytosis by alveolar epithelial cells, specifically type I cells. Under conditions of lung-particle overload, type II cells have also been implicated in the endocytosis and subsequent translocation of particles into the interstitium (Lehnert et al., 1992). Ultrafine particles (<~50 nm) have a much greater propensity to penetrate into the pulmonary interstitium and escape phagocytosis by alveolar macrophages (Ferin et al., 1991).

Following phagocytosis, the macrophages can migrate such that they are cleared via mucociliary flow; however, particle-laden alveolar macrophages are found in the lung up to many hundreds of days post-exposure, indicating that some macrophages do not migrate and that particles are re-ingested by other generations of alveolar macrophages.

Although mechanisms of clearance are very similar among different species, significant differences have been observed with respect to the pulmonary long-term retention of highly insoluble particles in different species, as summarized in Figure 10.5. Lung clearance of these particles in rats and hamsters is much faster than in dogs, guinea pigs and humans. Mice are also similar to rats in this regard. For the bulk of the deposited particles in the alveolar region, the clearance rate in the rat is ~1% per day, equivalent to a retention half-time of ~70 days. In contrast, the human pulmonary retention half-time for highly insoluble particles is ~500 days.

The change of clearance rate with time after cessation of exposure has been determined by Bailey et al. (1985) for humans. Based on their studies with radiolabelled fused alumino-silicate particles (1 and 4 µm in diameter) in humans, they found clearance to have two phases, with half-times of tens of days (intermediate phase) and several hundred days (slow phase). Clearance times were unaffected by particle size. Particle size did play an important role in alveolar macrophage-mediated lung clearance in rats instilled with ~10 µm polystyrene microspheres. Although they were phagocytized, they were not removed from the lungs of rats after a prolonged post-depositional period (Oberdörster et al., 1992b).

Figure 10.5 Long-term lung clearance of highly insoluble particles determined in different species by pulmonary retention measurements over several hundred days. (Adapted from Bailey et al., 1985b, 1989; Snipes et al., 1989)



There are a number of potential processes whereby particles are cleared via translocation through the interstitium. Phagocytosis by interstitial macrophages can occur, resulting in release of mediators and also transport of phagocytized particles in lymphatic channels to regional lymph nodes (Lehnert et al., 1992). Translocation of a small fraction of particles from lymph nodes into the blood compartment and then into any organ of the body may occur, as is suggested by results of studies with fibres that were found to appear in post-nodal lymph (Oberdörster, 1988) and by results with high lung burdens of titanium dioxide particles (Lee et al., 1985b) appearing in spleen and liver.

A recent report by Adamson et al. (1992) also indicates that transport of particle-containing interstitial macrophages back into the alveolar region may occur during a controlled inflammatory response. Particle size can play a decisive role in the ability of particles to translocate from the alveoli to the regional lymph nodes that receive lymphatic drainage from the lung. For example, Snipes and Clem (1981) found that 3 and 7 μm polystyrene particles instilled into the lungs of rats passed to the mediastinal lymph nodes, whereas larger (9 μm diameter) particles did not.

Rat studies have shown that a high interstitial particle load can result in an accumulation of particles in the bronchus-associated lymphatic tissue (BALT), which would be important for immune responses in the human lung (Ferin and Oberdörster, 1992). BALT has only recently been found to occur at significant levels in human lung (Pabst and Gehrke, 1990).

The retention and, accordingly, the effects of inhaled particles may also be influenced by the level of body activity. Exercise done by hamsters after they were acutely exposed to a colloidal gold aerosol (0.005-0.02 μm in diameter) has been reported to enhance the clearance of the particles from their lungs (Sweeney et al., 1990). Conversely, Collier et al. (1988) found that resting and exercising rats inhaling ^{57}Co-labelled fused alumino-silicate particles (~1-2 μm in diameter) showed no difference in the lung clearance of these particles.

Clearance mechanisms and pathways for soluble particle compounds involve diffusional transport via the tight junctions of the airway epithelium into underlying structures, including capillaries of the blood and lymph circulation. Pinocytotic processes (i.e., the cells engulf the particles within membrane vesicles) across the alveolar epithelium have also been described. Once in the circulatory system, transport to storage and excretory organs occurs as the final stage of the lung clearance process. Solubilization of particle compounds in the acidic milieu of the alveolar macrophage phagolysosome can also contribute significantly to dissolution of these particles. Additional details of lung clearance of inhaled particulate matter materials are described in several review articles (e.g., Schlesinger, 1985; Oberdörster, 1988; Snipes et al., 1989).

10.9.3 Conclusion

Quantitative extrapolation of results from animal studies to humans for purposes of risk assessment requires not only consideration of the differences in anatomical, morphological and functional aspects of the different respiratory tracts, but also detailed dosimetric adjustments to account for exposure-dose-response relationships obtained from animal studies. Dosimetric adjustments that must be made to appropriately extrapolate animal studies to humans would include adjustments for differences in particle deposition efficiencies in the respiratory tract and differences in retention and clearance kinetics. Deposition efficiencies of inhaled particles in the various regions of the respiratory tract differ between laboratory animals and humans. For example, respirable particles for humans can be larger than 5 μm, whereas for rats those particles are no longer respirable. Once the particles are deposited in the respiratory tract, they will be removed by specific clearance mechanisms. The main removal mechanisms for highly insoluble particles consist of the mucociliary escalator in the conducting airways and of phagocytosis by alveolar macrophages in the alveolar region. The particle removal rates vary considerably among species – e.g., the rat has a pulmonary retention half-time for highly insoluble particles of ~70 days, whereas in humans the retention half-time is in the order of 500 days. These species' differences need to be taken into account when interpreting or extrapolating results from animal inhalation studies.

An example of comparative dosimetry for particle exposure is illustrated by Miller et al. (1995). Their findings show that, while deposition of particles on a mass per unit alveolar surface area is not different between humans and rats, dose metrics based upon particle number per various anatomical parameters (ventilatory unit, alveolus, or alveolar macrophage) exhibit marked differences between rats and humans, particularly for particles 0.1-0.3 μm in size. Based on the calculations per ventilatory unit or per alveolus, humans receive much greater numbers of particles than do rats when exposed to the same concentration and size of particles. The trend of differences between humans and rats is even more pronounced for the individuals with compromised lungs (smokers, asthmatics and patients with chronic obstructive pulmonary disease) compared with normal subjects. Therefore, rats exposed to 1000 to 1500 $\mu g/m^3$ of particles may actually have received the levels of particles equivalent to 120-150 $\mu g/m^3$ in humans.

On the other hand, a purely dosimetric adjustment for extrapolation from experimental animals to humans will not be sufficient, as it is based on the unproven assumption that the same dose in the respiratory tracts of experimental animals and of humans will result in the same effect. Most likely, this is not the case. Even between rodent species, responses vary considerably; for example, pulmonary inflammatory and fibrogenic effects after inhalation of highly insoluble particles are most readily induced in rats, much less in mice and least in hamsters. Similarly, particle-induced lung tumours were seen only in rats but not in mice and hamsters. However, these particle-induced responses were seen only at inhaled concentrations of particles that led to very high lung burdens during chronic exposures (i.e., they are not relevant for extrapolation to humans).

Given these limitations, the animal toxicology studies should be interpreted in terms of how such studies might lead to an understanding of the mechanisms that lead to the causal path for the effects reported in the human epidemiological studies.

10.10 MECHANISTIC EVALUATION

The animal studies clearly show effects on the lungs resulting from the inhalation of particulate matter, effects that can be attributed to a particle effect rather than to the inherent chemical toxicity of the particle. These effects include decreased lung function, as measured by a number of parameters; decreased (or in some cases increased) particle clearance from the lung; increased alveolar macrophage numbers and activity (although the more cytotoxic particles can reduce both alveolar macrophage numbers and activity); decreased antimicrobial defence capacity (although this may be related to the cytotoxicity of the particle); biochemical changes associated with activation of alveolar macrophages or fibrosis; and morphological changes, including collagen formation and fibrosis, lipoproteinosis and epithelial hyperplasia. Cardiovascular abnormalities have also been observed after acute exposure to particulate matter. There is also evidence that prolonged exposure to high levels of particles can lead to lung cancer in rats.

A significant concern with all these studies is that the particle concentrations required to generate the toxic effects are well above those reported in the ambient environment. Many studies required particle levels that exceeded the clearance capacity of the lung to such an extent that clearance essentially shut down – the so-called "particle overload" (Morrow and Mermelstein, 1988; Morrow, 1994).

What these studies have demonstrated is that within the range of particles ≤10 μm in diameter (i.e., PM_{10}), the smaller particles seem to have more pronounced effects. This would not be unexpected if the total particle load was seen in terms of the number of particles, rather than particle mass. As shown in Figure 10.6, the urban ambient aerosol is represented by a trimodal particle size distribution, with an ultrafine, a fine and a coarse mode. Essentially all of the particles in these three different modes are respirable and would conform to the PM_{10} definition. However, some more recent epidemiological studies have suggested that the fine fraction of the PM_{10} particles (e.g., $PM_{2.5}$) correlates better with observed adverse effects.

Table 10.1 compares the number of particles per cubic centimetre of air for the same mass concentrations, for differently sized particles. As can be seen from this table, a particle with a diameter of 2.5 μm (i.e., conforming to the $PM_{2.5}$ definition) contributes only 1 particle/cm^3 to a total mass concentration of 10 $\mu g/m^3$, whereas it takes 2.4 million particles of 20 nm diameter (i.e., ultrafine particles) to achieve the same mass concentration. In addition, particle surface area is also considerably increased for the fine and ultrafine particles.

As for mechanisms, it has been suggested that the ultrafine particles, by virtue of their numbers and level of toxicity, could present a potential hypothesis to explain the effects reported at low total particle mass levels. Unfortunately, ambient monitoring of the ultrafine particle mode of the urban aerosol is much more difficult, and, therefore, few data are available.

The exact mechanism of the increased toxicity of ultrafine particles is not known. However, several factors appear to be important. One is that their

Figure 10. 6 Particle volume distribution of ambient aerosol measurement by Wilson et al., (1977) in road tests. A typical trimodal distribution showing a coarse, an accumulation (fine), and a nuclei (ultrafine) mode is shown with geometric mean diameter of 4.9 µm, and 0.21 µm and 18 nm, respectively. (SC=geometric standard deviation). (Adapted from Wilson et al., 1977)

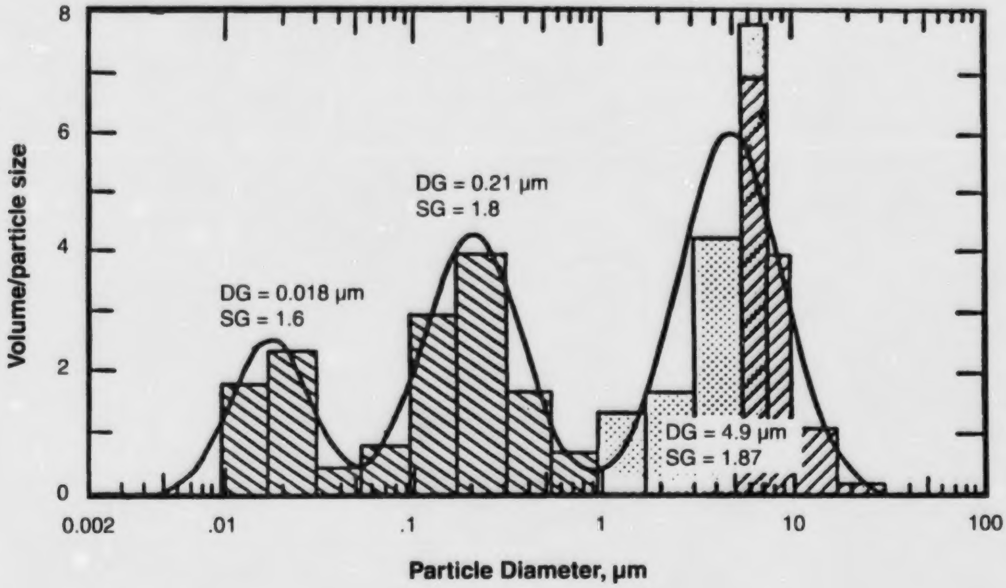


Table 10.1 Numbers and Surface Areas of Monodisperse Particles of Unit Density of Different Sizes at a Mass Concentration of 10 µg/m^3 (Oberdörster et al., 1995)

Particle diameter (µm)	Particle no./cm^3 air	Surface area (µm^2/cm^3 air)
0.02	2 400 000	3 016
0.1	19 100	600
0.5	153	120
1.0	19	60
2.5	1.2	24

considerable surface area represents a larger area for interaction with biologically active sites; the surface area of retained particles of different sizes, including ultrafines, was found to correlate with the pulmonary inflammatory response (Oberdörster et al., 1992). Another factor is that such ultrafine particles are not as readily phagocytized by alveolar macrophages, and therefore they are more likely to react with alveolar epithelial cells and be translocated to the interstitium (Ferin and Oberdörster, 1992). Finally, it appears that inhalation of ultrafine particles as singlet particles, especially when freshly generated, is of significance and leads to greater effects than inhalation of aggregates of ultrafine particles (Oberdörster et al., 1994a). The highly reactive surfaces of these particles apparently lead to severe damage of the alveolar-epithelial barrier, causing acute pulmonary edema and inflammation, although a pathogenic mechanism is still to be elucidated.

No firm conclusions can be drawn from the results of the numerous animal toxicology studies described in this chapter to answer the question of which particle type and size is most likely to cause the adverse effects observed in human epidemiological studies, described in Chapter 12. With respect to increased morbidity in children due to respiratory tract symptoms (e.g., Dassen et al., 1986; Burnett et al., 1995), it is plausible that acidic aerosols (i.e., sulphates) could be a major causative factor. Results from animal studies, almost exclusively in guinea pigs, have unequivocally shown that these aerosols lead to increased bronchial reactivity that may be of particular importance for asthmatic individuals, although pure sulphate aerosols had to be administered at >100 µg/m^3 to cause these effects (Chen et al., 1992b). The mean concentrations of ambient sulphates measured across Canada from 1984 to 1993 ranged from 1.6 to 6 μg/m^3 (Dann, 1994), and increased bronchial reactivity at these levels has not been observed in animal studies. However, Chen et al. (1992a) showed that sulphuric acid exposure levels as low as 20 and 30 μg/m^3, when the acid was coated onto carrier particles, caused increased hypersensitivity in guinea pigs. The carrier particles were generated as ultrafine particles (50 nm), which had a high specific surface area (Table 10.1) and, therefore, could well function as carriers for adsorbed compounds. As the co-administered ultrafine carrier particles were at a concentration of ~1-2 mg/m^3, they were in all likelihood aggregated. Still, an additional effect of these particles cannot be excluded; moreover, these high levels of particle air pollution do not occur in the ambient environment.

Thus, although the particle carrier hypothesis appears to be reasonable, the much higher experimental concentrations compared with relevant ambient exposure levels make it very difficult to use such data for direct extrapolation to humans. Regardless, it appears that the fine-particle phase is more important than the larger particles for these types of effects.

The role of surface-complexed iron (Fe^{3+}) on particles for inflammatory responses in the respiratory tract should also be considered (Ghio et al., 1992; Ghio and Hatch 1993; Costa et al., 1994). An increase in reactive oxygen species via the Fenton and Haber-Weiss reaction may potentiate the inflammation. However, concentrations of iron needed to cause the increased inflammatory response as well as the necessary concentrations of inhaled particles are not likely to be encountered at normal ambient particle levels.

The epidemiological finding that ambient particulate matter air concentrations below 100 μg/m^3 are associated with increased mortality (Section 12.2) cannot be directly explained by animal studies as far as PM_{10} and $PM_{2.5}$ particles are concerned. Except for the ultrafine fume particles, none of the other particle types and sizes used in animal inhalation studies cause such acute dramatic effects, including high mortality at relatively low concentrations. However, one comparative dosimetric analysis conducted by Miller et al. (1995) indicates that, based on the calculations per ventilatory unit or per alveolus, humans receive much greater numbers of particles than do rats when exposed to the same concentration of PM. The trend of differences between humans and rats is even more pronounced for the individuals with compromised lungs (smokers, asthmatics and patients with chronic obstructive pulmonary disease) compared with normal subjects. Therefore, rats exposed to 1000 to 1500 μg/m^3 of particles may actually have received the levels of particles equivalent to 120-150 μg/m^3 in humans.

The mechanism for particle-induced cardiovascular response is not yet clear. It may involve the oxidation of low density lipoprotein by reactive oxygen species accompanying particulate matter pollution. Oxidized low density lipoprotein has been found to cause endothelial damage, the proliferation of smooth-muscle cells, monocyte-endothelial interactions, platelet aggregation, and inhibition of endothelial-derived relaxation of vascular smooth muscles (Morel et al., 1983; Ocana, 1989; Berliner et al., 1990; Cushing et al., 1990; Ezaki et al., 1994; Ikeda et al., 1995; Ichinose et al., 1995). Thus diesel exhaust particle-induced oxidization of low density lipoprotein may play a pathological role in inducing atherosclerosis and coronary vasospasm.

With respect to an association of particulate matter air pollution with increased lung cancer (Section 11.4.6), results of animal studies are again not very informative. Animal inhalation studies aimed at investigating carcinogenic potentials of inhaled particles have shown that high inhaled concentrations of diverse particles such as titanium dioxide, carbon black and diesel exhaust cause lung tumours in rats. Initially, it was thought that diesel exhaust-induced lung tumours were due to the existence of PAHs adsorbed onto the diesel particles (Heinrich et al., 1986a); however, subsequently performed studies with carbon black showed that these particles without adsorbed PAHs were as effective in inducing lung tumours (Heinrich et al., 1992; Pott et al., 1994).

Thus, although particles in general have been shown to induce lung tumours in rats, inhaled concentrations were in the milligram per cubic metre range, far beyond current ambient environmental levels. Indeed, these high experimental exposure concentrations have been shown to lead to particle overload-related reactions in the lung, which include chronic inflammatory and fibrotic responses (Morrow and Mermelstein, 1988; Morrow, 1994). The term "overload" indicates that the alveolar macrophages have been overwhelmed to such an extent that their ability to clear particles out of the lung becomes impaired. This impairment of clearance in turn results in increasing amounts of particles being retained in the lung, with subsequent translocation to interstitial sites and respective effects on alveolar, epithelial and interstitial cells. It has even been shown that DNA adducts are induced by high levels of carbon black as well as diesel soot in the lungs of rats, although the quality of the adducts may differ between the two particle types (Bond et al., 1990). Mechanistically, it is suggested that the chronic inflammatory response occurring in the lung from high particle load leads to the release of many mediators – i.e., cytokines, growth factors, enzymes and oxygen-derived radicals – from alveolar macrophages and possibly other lung cells (Driscoll et al., 1990; Morrow, 1994). These mediators in turn induce increased epithelial cell proliferative responses and adduct formation, thereby increasing the possibility of mutations becoming manifest, and they may eventually result in tumorigenesis.

This "overload" concept, and its implications with respect to the interpretation of results from studies conducted at high particle loads, is similar to the maximum tolerated dose (MTD) concept in cancer bioassay protocols. Without going into the specific details of the MTD concept, the basic premise is that in many cancer bioassay protocols, including the National Toxicology Program (NTP), the high-dose group in the study should be dosed at a level that induces some level of toxicity. Concerns have been raised in the scientific community that such dose levels may not be relevant, as they may exceed the normal ability of the test animals to detoxify the material or cause tissue damage that may lead to secondary processes that would not be seen at lower, more environmentally relevant, dose levels. Many scientific papers have been written on the MTD issue, too many to cite adequately; however, an excellent critique written by Haseman (1985) provides a basic overview of the issues.

Thus, although the hypothetical, mechanistic scenario derived from the animal studies may be very plausible, these high-load chronic inhalation studies performed in experimental animals may have no relevance for low-level ambient exposures.

Complex interactions between differing constituents of air contaminants have been examined in animal studies only to a limited degree, mostly focussing on one particle and one gas-phase compound only. As shown in Section 10.9, such combined exposures resulted in mixed responses, showing either no effect of the combination or some synergism with respect to certain endpoints, but overall the results are equivocal. However, realistic environmental conditions are far more complex than those re-created in the laboratory, and the respective effects of exposures to such mixtures are not well predictable. In addition, it should be kept in mind that susceptible groups within the population may react much more sensitively to a given inhaled particle/gas mixture. Furthermore, a compromised respiratory system will also be much more sensitive to the adverse effects of inhaled pollutants. Unfortunately, most of the studies performed today have been concerned with the deposition and clearance efficiencies in such conditions, rather than with health endpoints *per se*.

10.11 CONCLUSIONS

The data available from experimental animal studies indicate that the particle types found in the ambient atmosphere that are most likely to induce acute adverse effects include the fine particle mode, including acidic sulphates, possibly occurring as a coating on fine or even ultrafine carrier particles. These effects include decreased lung function, decreased (or in some cases increased) particle clearance from airways, altered alveolar macrophage numbers and functions, modified immunological function, cytotoxicity and histological changes, and cardiographic abnormalities. There is also evidence that prolonged exposure to high levels of particles can lead to lung cancer in rats. Bronchial hypersensitivity to non-specific stimuli and increased morbidity from cardio-respiratory symptoms would most likely occur in animals pre-disposed to cardio-respiratory diseases.

It appears that the ultrafine particle mode may be of significant toxicological importance. On the other hand, the coarse particle mode is less likely to induce acute adverse responses, although these larger particles may well contribute to such effects. The particle deposition data clearly indicate that TSP is not an appropriate measure of particulate matter as it relates to human health effects, as only particles <10 µm in diameter (and possibly up to 15 µm in diameter during mouth breathing) penetrate to the lungs. The evidence from the animal toxicology studies indicates that the toxic effects of airborne particles are more likely to be associated with the fine mode of particulate matter air pollution (i.e., $PM_{2.5}$, as opposed to PM_{10}), including acidic sulphates, possibly occurring as a coating on fine or even ultrafine carrier particles.

The interaction between ozone and sulphuric acid evidently is one in which the damaging effects of the ozone in the centriacinar region of the lung are enhanced by sulphuric acid aerosols.

11 HUMAN HEALTH EFFECTS – CLINICAL STUDIES

A series of recent reports have shown that variation in ambient PM_{10} concentrations, even at current levels, is positively associated with the variation in daily cardiopulmonary mortality and total mortality, excluding accidental and suicide deaths (Bascom, 1994). These associations have been shown in different communities, including Philadelphia, St. Louis, the Utah Valley and Santa Clara County, California of the US, and in Ontario, Canada, with widely differing particle composition and climates. Despite these epidemiological findings for acute and chronic adverse health effects from air pollution associated with respirable particles, there are as yet few complementary data from animal toxicology studies and controlled human exposures to respirable particles. In this chapter, a review of controlled human exposure studies (clinical studies) relevant to understanding the effects of inhaled particulate matter at current concentrations is presented, based on a contract report prepared for Health Canada (Utell and Samet, 1994).

11.1 CLINICAL STUDIES

Carefully controlled, quantitative studies of exposed humans offer a complementary approach to epidemiological investigations (Utell et al., 1993). Human clinical studies utilize laboratory atmospheric conditions considered relevant to ambient pollutant concentrations and document the health effects that result from breathing known concentrations of pollutants. Advantage is taken of the highly controlled environment to identify responses to individual pollutants and characterize an exposure-response relationship. In addition, such a controlled environment provides the opportunity to examine interactions among pollutants or with other environmental variables, such as exercise, humidity or temperature. Insofar as individuals with acute and chronic respiratory diseases can participate in exposure protocols, potentially susceptible populations may also be studied. This approach also has its limitations. For practical and ethical reasons, studies must be limited to small groups, which may not represent larger populations. Exposure must also be limited to short durations and to concentrations of pollutants expected to produce mild and transient responses. Exposures are often limited to a single pollutant, or to a very limited pollutant mix that never replicates the complex mixture to which populations are actually exposed. An endpoint assessment will invariably include pulmonary function which may only indicate physiological reflex rather than tissue injury. Furthermore, transient responses in clinical studies have never been validated as predictors of more chronic and persistent effects.

An important feature of controlled clinical studies is the opportunity to examine both healthy volunteers and individuals with underlying cardiopulmonary diseases who are likely to be particularly vulnerable to particulate pollution. Subjects are typically characterized by age, gender, race and lung function. Normal volunteers are characterized by the absence of allergies, often documented by skin testing and lack of hyperreactive airways as assessed by inhalation challenge tests. Within the rubric of healthy volunteers (absence of allergies and normal lung function), adolescent, elderly normal and "smoker" subgroups have been studied in various protocols. Healthy volunteers are typically able to perform vigorous exercise for extended periods and are usually able to tolerate the more invasive investigative techniques such as bronchoalveolar lavage (BAL).

Other "susceptible" subpopulations recruited to participate in clinical studies include asthmatics, individuals with allergies or acute upper respiratory infections and subjects with chronic obstructive pulmonary disease (COPD) or coronary artery disease. For research and clinical purposes, asthmatics can be characterized by their responsiveness to methacholine or carbachol, presence or absence of allergy (skin tests or IgE levels), use of medications, severity of symptoms and degree of airway obstruction assessed by pulmonary function tests. Severely obstructed asthmatics have rarely participated in controlled clinical studies. In Table 11.1 some potential links between susceptible groups and morbidity effects are outlined.

Table 11.1 Potential Links Between Susceptible Groups and Morbidity Effects

Population	Adverse health effects
COPD and asthma	worsening of V/Q
— exacerbation	increased airway reactivity
— pulmonary edema	increased work of breathing
— respiratory infection	increased risk of respiratory infection
individuals with respiratory infections	
— exacerbation	worsening of V/Q
— hypoxemia	hypoxia
individuals with cardiac disease	
— arrhythmia	worsening of V/Q
— pulmonary edema	hypoxia
	pulmonary hypertension

Abbreviations: COPD = chronic obstructive pulmonary disease
V/Q = ventilation-perfusion ratio

Exercise done on either a treadmill or a bicycle ergometer is generally an important component of exposure studies. Exercise enhances the pollutant dose both by increasing ventilation and by causing a switch from nasal to oral breathing. As described in the proceeding chapter, oral breathing effectively bypasses the nose which would otherwise filter out particles and gases, therefore increasing the delivery of particles to the lower respiratory tract. In addition, the effect of exercise on airway drying may enhance the response to pollutants. Minute ventilation during exposure is clearly an important determinant of the magnitude of change in selected physiological measures. Less clear is the influence of continuous versus intermittent exercise on pulmonary function responses. The impact on pulmonary function of exercising at different intensities during pollutant exposures has been reviewed by Horvath (1985).

Finally, the double-blind protocol, which uses a cross-over design, with a sodium chloride control exposure for the aerosol and a clean air control exposure for the gaseous pollutant, is optimal. In such experiments, the investigator, the technician and the subject are unaware of the exposure conditions, that is, whether the exposure on a single day involves a pollutant or control substance. Such a strategy eliminates potential observer bias and avoids relaying clues to volunteers regarding anticipated responses. This is especially important when studying asthmatics, as a significant number of asthmatics may tend to respond to psychological stimuli with bronchoconstriction (Spector et al., 1976).

11.2 SOLUBLE PARTICLES – ACIDIC AND SULPHATE

As described thoroughly in Chapter 3, particulate air pollution is a complex mix of chemical species, of both natural and anthropogenic origin. Of this mixture, clinical studies have tended to examine only the latter category (anthropogenic particles), especially inorganic nitrates and sulphates, since these have played a prominent role in "acid rain." These studies were reviewed previously by Utell (1985) and more recently by the US EPA's Acid Aerosols Issue Paper (US EPA, 1989).

In the atmosphere, the major acid sulphate species include $(NH_4)_2SO_4$, NH_4HSO_4 and H_2SO_4. In the laboratory, pulmonary responses of normal and asthmatic subjects have been assessed following exposure to each of these acids. In this section, a summary of recent controlled human exposure studies involving acidic sulphates is presented. Nitrate particles are not considered in this review, as they have not been found to invoke symptoms or functional decrements (US EPA, 1989), or cellular and biochemical responses in airway lavage fluids (Aris et al., 1993) in clinical studies at concentrations below 1000 µg/m^3. The few reports of controlled exposures to diesel, carbon black or other particles will also be reviewed. Studies in individuals with underlying lung disease (i.e., "susceptible subpopulations") are emphasized, recognizing that responses are typically defined by changes in mechanics; however, findings from healthy volunteers, especially alterations in defence mechanisms or cellular responses, are also considered, if relevant.

11.2.1 Studies of Healthy Volunteers

Controlled experimental exposures have been used to examine the respiratory effects resulting from the inhalation of acidic aerosols, with measured outcomes limited primarily to symptoms and airway function. Although exposures to concentrations of H_2SO_4 aerosols below 1000 µg/m^3 generally do not alter airway function of normal subjects (Utell et al., 1984), Utell et al. (1983a) observed an increase in throat irritation and airway reactivity to carbachol inhalation in normal volunteers 24 hours after exposure to 450 µg/m^3. No change in airway reactivity was detected immediately after exposure. Effects of H_2SO_4 on slowing of mucociliary clearance in small airways begin at concentrations as low as 100 µg/m^3; the duration of the slowing increases with hours of exposure (Spektor et al., 1989).

Only one study has been done to examine the effects of H_2SO_4 aerosols on host defence mechanisms at the alveolar level (Frampton et al., 1992). Non-smoking volunteers were exposed for two hours to an aerosol of approximately 1000 µg/m^3 H_2SO_4 or to a control substance (sodium chloride, NaCl), with intermittent exercise, in a random double-blind fashion. BAL was done 18 hours after exposure to detect evidence of an inflammatory response, change in alveolar cell subpopulations or changes in alveolar macrophage function, all components of host defence. Compared to exposure to NaCl, exposure to H_2SO_4 did not increase numbers of polymorphonuclear leucocytes (PMN) in BAL fluid. The percentage of T lymphocytes decreased in association with H_2SO_4 exposure, but the decrement was not statistically significant. Antibody-mediated cytotoxicity of alveolar macrophages increased in association with H_2SO_4 exposure. Significant changes were not found in release of superoxide anion or inactivation of influenza virus *in vitro*. The investigators concluded that brief exposures to H_2SO_4 aerosols at 1000 µg/m^3 did not cause an influx of inflammatory cells into the alveolar space. No evidence was found for alteration in alveolar antimicrobial defence 18 hours after exposure.

11.2.2 Studies of Asthmatic Volunteers

As with SO_2, asthmatic subjects have been found most susceptible to the effects of acidic aerosol exposure, although different laboratories have found differing threshold exposure concentrations. Generally, however, studies of adult asthmatics have failed to show alterations in lung function at levels below 200 µg/m^3. Utell et al. (1983b) exposed asthmatics to H_2SO_4, NH_4HSO_4, sodium sulphate hydrogen ($NaHSO_4$) and a control NaCl aerosol at concentrations of 100, 450 and 1000 µg/m^3. Following exposures to the 450 and 1000 µg/m^3 aerosols for 16 minutes, specific airway conductance decreased in relation to the acidity of the aerosol, supporting the hypothesis that airway effects are related to acidity rather than to the sulphate ion. More prolonged exposures to H_2SO_4 aerosols have also been performed. With 10-minute exercise periods every 30 minutes, a two-hour exposure to 100 µg/m^3 H_2SO_4 aerosol resulted in a small reduction in flow rates. Response to H_2SO_4 was highest after the first 45 minutes, then airway function recovered, returning towards the baseline, indicating that the effect did not seem progressive over time. Respiratory ammonia was identified as a factor influencing response to H_2SO_4 aerosols. Utell et al. (1989) enhanced airway responses to inhalation of 350 µg/m^3 aerosols in exercising asthmatics by reducing the level of endogenous respiratory ammonia. This provided further evidence that aerosol acidity significantly affected bronchoconstriction caused by inhalation of sulphate aerosols.

Adolescent asthmatics appear to be more sensitive than adult asthmatics to the effects of acidic aerosols. Functional decrements have been observed in adolescent asthmatics at levels as low as 35 µg/m^3 (MMAD 0.6 µm) for 40-90 minutes (Koenig et al., 1989; Hanley et al., 1992; Koenig et al., 1992). The low range of H_2SO_4 concentration is around the peak values measured during the summer months in the eastern United States and southern Canada. The apparent difference in sensitivity of adult and adolescent asthmatics may also be due to selection bias, differences in aerosol sizes or exposure protocols. However, adolescent asthmatics respond to concentrations of H_2SO_4 aerosols an order of magnitude lower than those causing a response in normal subjects. Field studies in summer camps of both normal and asthmatic children reported decrements in pulmonary function during pollution episodes that included increased levels of acidic aerosols (Raizenne et al., 1989). This reinforces the concern that children and adolescents may be particularly susceptible to the effects of acidic atmospheres.

The sensitivity of asthmatics to acidic aerosols has also been observed in elderly subjects aged 60-75 years (Koenig et al., 1993). When elderly normal and asthmatic volunteers inhaled H_2SO_4 or $(NH_4)_2SO_4$ (70 µg/m^3, MMAD 0.6 µm) for 40 minutes with mild exercise, a significant increase in total respiratory resistance was seen in the asthmatic subjects but not in normal subjects. However, compared with younger asthmatic subjects under a similar H_2SO_4 dose regimen (Koenig et al., 1992), elderly asthmatic subjects do not seem more at risk for adverse respiratory effects from inhalation of acid aerosols.

In another publication by Koenig et al. (1990), asthmatic adolescents were preexposed to ozone (0.12 ppm, 45 min) followed by a low level of SO_2 (0.1 ppm, 15 min) with intermittent exercise ($\underline{V}_E$ = 30 L/min). Preexposure to ozone elicited a greater degree of bronchial responsiveness than did pure air followed by SO_2, or two exposures to ozone alone, indicating some synergism between the effects of SO_2 and ozone. Given that both ozone and SO_2 concentrations were at subthreshold levels for the experimental design used, it is tantalizing to speculate that preexposure to ozone would likely potentiate exposures to acid aerosols as well.

Frampton et al. (1995) found that preexposure to H_2SO_4 may potentiate ambient levels of ozone-induced health effects in allergic asthmatic subjects (28 ± 7 years old, n = 30). In this study, each healthy (28 ± 5 years old, n = 30) and asthmatic subject was preexposed to 100 µg/m^3 H_2SO_4 (MMAD 0.66 µm) or NaCl aerosols (MMAD 0.45 µm), for 3 h. One day later (24 hours), the subjects were exposed to 0.08, 0.12, or 0.18 ppm ozone for 3 h. During each exposure, subjects performed intermittent exercise ($\underline{V}_E$ = 33 - 40 L/min). In healthy subjects, there was no convincing evidence for symptomatic or physiologic effects of exposure to either NaCl or H_2SO_4 aerosols or ozone alone, and there was no clear evidence for an effect of aerosol preexposure on the ozone response. In asthmatic patients, ozone (starting from 0.08 ppm), NaCl and H_2SO_4 aerosols independently caused decrements of forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1); yet the severity of effects induced by NaCl and H_2SO_4 aerosols were not significantly different. H_2SO_4 aerosol altered the pattern of spirometric response to ozone in comparison with NaCl preexposure and appeared to enhance the small mean decrements in FVC and FEV_1 that occurred in response to 0.18 ppm ozone.

Concurrent exposure of healthy and asthmatic adults to low levels of ozone (0.12 ppm) plus sulphuric acid aerosols (100 µg/m^3) did not cause substantial changes in lung function and symptoms than exposure to ozone alone, in either asthmatic or healthy subjects (Linn et al., 1994). In a very recent study conducted by Linn et al. (1997), current exposure of children with allergy or asthma (n = 26) to pollutants (containing H_2SO_4 aerosol 100 µg/m^3, ozone 0.1 ppm, and SO_2 0.1 ppm) for 4 hours had a significantly positive association with symptoms, while in healthy children that association was not significant. The pollutant mixture did not significantly alter pulmonary function parameters in either groups (Linn et al., 1997).

11.2.3 Volunteers with COPD

Based on both pathophysiologic considerations and epidemiological observations, subjects with COPD are considered potentially at high risk for mortality and morbidity from particles. To determine whether low-level H_2SO_4 aerosol inhalation induced alterations in lung function in volunteers with COPD, older subjects (mean age = 62 years) were exposed to 90 µg/m^3 H_2SO_4 aerosols in an environmental chamber for two hours (Morrow et al., 1994). Subjects were defined by dyspnea on exertion, obstructive airway disease (FEV_1 = 1.4 L or 53% of predicted, and FEV_1/FVC = 0.56) and a lack of response to bronchodilators. In contrast to findings in asthmatic patients, the volunteers with COPD showed no greater decrements in pulmonary mechanics in response to H_2SO_4 than to the control aerosol, NaCl. Subjects with COPD, presumably the most vulnerable subpopulation, showed virtually no change in flow rates, even with periods of intermittent mild exercise during their exposure periods. Therefore, in the only study evaluating responses to specific components of particulate matter, enhanced responsiveness to acid could not be identified. This cannot be taken to mean that they might not respond to other forms of particulate matter.

11.2.4 Summary of Results on Soluble Particles

The findings of these studies provide evidence that normal individuals experience few or no adverse effects on airway function or on defence or immunological mechanisms, at relatively high concentrations of acidic particulate pollution compared to ambient levels. However, acidity has been shown to affect the slowing of mucociliary clearance at concentrations as low as 100 ug/m^3. Asthmatic patients may experience adverse effects on airway function in clinical studies at concentrations equivalent to relatively high ambient levels. These effects are more pronounced in adolescents and children, at concentrations experienced on occasion in ambient air (~ 35 µg/m^3 H_2SO_4 for 40 min). Collectively, these data demonstrate that asthmatics do manifest significant, although small, reductions in their performance during moderate physical activity after inhaling acidic aerosol concentrations close to high ambient levels. For asthmatics, the irritant potency of sulphate aerosols appears related to acidity *per se*. Inhalation of the more acidic sulphates produces the most significant bronchoconstriction. Exposure to less acidic sulphate aerosols causes no significant change in lung function. Other findings suggest that the amount of titratable acidity in an aerosol is a determinant of pulmonary effects (Fine et al., 1987). In any case, the decrements in lung function are relatively small after exposure to acid particles and are not progressive, and they are mitigated by ammonia neutralization. There exists evidence that preexposure to H_2SO_4 may potentiate ambient levels of ozone-induced symptoms and pulmonary function decrements in asthmatic subjects, and that a concurrent exposure of asthmatic children to both pollutants may also potentiate symptoms, but not pulmonary function decrements. The individuals with the most severe obstructive disease, namely COPD, do not appear to respond to acid particle inhalation with bronchoconstriction or symptoms thereof.

11.3 INSOLUBLE PARTICLES

11.3.1 Deposition and Clearance Studies

Inhalation of insoluble particles has been used primarily as a tracer to examine deposition and long-term clearance kinetics from the human lung, not for a determination of inherent toxicity. In a study reported by Bailey et al. (1982), subjects inhaled 10 breaths of 25 µL of fused alumino-silicate particles in ethanol. Monodisperse 1.2 µm- and 3.9 µm-diameter particles were labelled with radioactive tracers. Clearance was followed for more than 200 days. Approximately 8% of 1.2 µm and 40% of 3.9 µm particles cleared within six days. Overall retention of the remaining material showed half-times averaging 320 days, with considerable inter-subject variation. Approximately 10 other studies examining long-term clearance rates in humans have been conducted with particles such as iron oxide, manganese oxide and polystyrene; clearance half-times have generally been faster than 320 days (Bailey et al., 1982). No effects on symptoms or lung function changes were noted in these studies, presumably because of the low particle concentrations and very short exposure times.

Studies of deposition of diethyl hexyl sebacate particles of 0.02 to 0.4 µm has been examined in subjects with obstructive and restrictive lung diseases (Anderson et al., 1990). Particle loads were achieved after 4-10 breaths, and inhalation of the experimental aerosols probably required less than one minute. The data show that deposition of these ultrafine particles was increased in subjects with obstructive lung disease when compared with healthy subjects, while it was unchanged in subjects with restrictive lung disease. The lung function tests were not done. The absence of clinical responses was undoubtedly related to limited exposure (4-10 breaths) of the aerosol as well as small sample sizes.

11.3.2 Effects on the Respiratory Tract

Few studies have been done on insoluble particles and their effects on the human respiratory tract. However, in one recent controlled exposure study, healthy and asthmatic volunteers inhaled respirable carbon particles (Anderson et al., 1992). Four exposure scenarios were defined: (1) clean air; (2) 0.5 µm H_2SO_4 aerosol at 100 µg/m^3; (3) 0.5 µm carbon aerosol at 250 µg/m^3; and (4) 250 µg/m^3 carbon plus 100 µg/m^3 H_2SO_4 aerosol generated from fuming H_2SO_4. Electron microscopy showed that nearly all the acid in group 4 became attached to carbon particle surfaces and that most particles remained in the submicrometre range. All exposures were for one hour, with alternate 10-minute periods of exercise and rest. Respiratory symptoms and lung function were measured before, during and after exposure, whereas bronchial reactivity to methacholine was measured post-exposure. Analyses were done for effects of H_2SO_4 or carbon, separate or interactive, on all outcome measures. The investigators concluded that for both healthy and asthmatic subgroups, the associations between mean changes in lung function measures and the experimental pollutants were small and of no clinical significance. In persons without lung disease, forced expiratory function measurements did not show any significant variations associated with carbon or H_2SO_4. Likewise, the changes in lung function for the asthmatic group as a whole did not suggest a significant adverse effect of the pollutants, separately or combined, in comparison with the effect of clean air. However, one asthmatic subject showed severe clinical manifestations and substantial decrements in FEV_1 and FVC following carbon and acid treatments. Acid-coated carbon particles induced much greater adverse health responses than did acid or carbon black alone in this asthmatic subject, who might represent a vulnerable subgroup.

Finally, Sandstrom and Rudell (1991) examined the bronchoalveolar inflammatory response to inhalation of diesel exhaust. Diesel exhaust from an idling diesel engine was diluted with air and introduced into an exposure chamber. Median concentrations in the breathing zone were 3×10^6 particles/cm^3, 3.7 ppm nitric oxide (NO), 1.6 ppm NO_2, 27 ppm carbon monoxide (CO) and 0.3 mg/m^3 formaldehyde. Exposures were done for one hour and included moderate exercise on a bicycle for 10-minute periods alternating with rest. Lavage was done 18 hours after exposure and demonstrated an increase in neutrophils in the bronchoalveolar but not the bronchial portion of the lavage. Phagocytosis of opsonized yeast cells *in vitro* by macrophages from the BAL was significantly altered. As exposures to pollutants such as SO_2 or NO_2 alone have typically not induced an acute inflammatory response, the investigators speculated that the responses may have been caused by particles or hydrocarbons in the diesel exhaust.

Unfortunately, far too few clinical studies have been done with either carbon or diesel particles to allow meaningful conclusions to be drawn.

11.4 OVERALL CONCLUSIONS FROM CLINICAL STUDIES

Controlled human exposures to acidic and inert particles have not caused significant alterations in respiratory function in healthy individuals at relatively high levels compared to those generally experienced in the environment. The clinical studies identify asthmatics as a susceptible population, but not persons with chronic obstructive pulmonary disease (COPD) or the elderly, at least not for acidic particles, based on a single study. Asthmatics, especially children and adolescents, may experience adverse effects on airway function at concentrations experienced on occasion in ambient air (~ 35 µg/m^3 H_2SO_4 for 40 min).

None of the human clinical studies have used particle generation systems that reflect the complexity of ambient particles. Based on the extremely limited clinical database available on various species of particles, acidic aerosols produce the most significant bronchoconstriction, while the toxicity of sulfate is related to acidity *per se*. The toxicity of nitrates was not considered, since previous work had shown it not to exert effects on lung function at concentrations below 1000 µg/m^3 in clinical studies. Inert particles did not appear to affect lung function in either healthy or asthmatic volunteers in the few studies available. Although very fine particle diesel exhaust affected neutrophil production and macrophage clearance of microorganisms from the lung, the effects cannot be ascribed with certainty to particles, since formaldehyde and other combustion gases were also elevated.

Very little work has been carried out on the effect of particle size specifically on airway mucociliary function. Although limited studies have shown that fine particles (less than 2.5 µm) are cleared from the lung more slowly than larger particles, and that submicrometre (<1.0 µm) particles clear very slowly, taking more than one to two years in a few cases, especially in patients with obstructive lung diseases. A newly published study by Peters et al. (1997) shows that symptoms and decrements of peak expiratory flow in asthmatic subjects (n = 27) were significantly associated with the 5-day mean of the number of ultrafine particles (MMAD 0.01 to 2.5 µm)(Peters et al., 1997).

Evidence of a dose-response relationship, and increased risk of an adverse effect associated with increased exposure, bolsters the argument for causality. Although the ranges of particle concentrations usually exceed those experienced by the general population, little evidence for a dose-response relationship has been documented in the clinical toxicological literature. Even at high particle concentrations in susceptible subpopulations, acidic aerosols have been found to produce only small decrements in lung function.

Overall, the clinical data lend only very limited support to the observations seen in the epidemiology studies, particularly to the observations that high ambient particulate concentrations are associated with mortality within hours or a few days at most. They do implicate one susceptible subpopulation, asthmatics, who currently comprise five to eight percent of the population, a percentage that has been rising in the past decade in Canada as well as in other western countries. Possible explanations for the discrepancy between clinical and epidemiological data may lie in the following: (1) the experimental subjects can only be exposed to the tested air pollutants for short duration for practical and ethical reasons, while an urban pollution episode usually lasts a few days for general population exposure; a clinical study has shown that doubling the length of exposure to H_2SO_4 exerted greater effect on bronchial mucociliary clearance than did an order of magnitude increase in the concentration of H_2SO_4 (Spektor et al., 1989); (2) ethically investigating responses in those people most likely to be affected by air pollutants is almost impossible; (3) the pulmonary function parameters that are most often used in clinical studies may not be sensitive enough to indicate particle-induced adverse health effects; (4) artificial particles used in exposure chambers may not reflect the potential synergistic effects of particulate matter and aerosol mixtures; (5) in most human studies, the sizes of aerosols used are above 0.5 µm. Since nanometre-sized ultrafine particles have been found in animal studies to induce acute pulmonary inflammation and death at very low concentrations, and they are present in ambient air, ultrafine particles may be a good candidate to provoke acute alveolar inflammation with release of mediators capable, in susceptible individuals, of causing cardio-respiratory responses.

12 HUMAN HEALTH EFFECTS – EPIDEMIOLOGICAL STUDIES

It has generally been accepted since the 1970s that there is an association between respiratory health and high levels of particulate air pollution. Early studies focussed on severe pollution episodes, including the Meuse Valley, Belgium, in December 1930; the Manchester and Salfard area, England, 1931; Donora, Pennsylvania, 1948; London, England, 1952; as well as New York City, at approximately the same time. Although the technology of the day has limited the accuracy of reported levels, the associations observed between morbidity/mortality and these episodes of elevated particulate concentrations indicate a clear link between air pollution and adverse health effects. What has not been clear is whether these adverse effects do in fact occur at the much lower concentrations of air pollutants that are experienced today in North America and Western Europe. What has also not been clear is whether these effects were due to air pollution as a whole, or solely to particulate matter, and if so, to which species of particle, or whether the effects were related to one or more other air pollutants which are always present along with particles.

12.1 DESIGN ISSUES

12.1.1 Particle Metrics

Historically, air pollution studies have used different types of measures of particulate matter. One of the commonest of these has been total suspended particulates (TSP); this measure has been superseded in some countries (including Canada, the US, Britain, and some western European countries) by more physiologically-relevant measures of inhalable and/or respirable particles. The TSP method of measurement collects material up to 27-45 μm aerodynamic diameter (d_a) over a 24 h period for assessment by direct weighing. PM_{10}, $PM_{2.5}$, and other less frequently used size selective methods are also gravimetric. Other types of measurements commonly used are the British Smoke Shade or Black Smoke (BS) method and Coefficient of Haze (CoH), which were discussed in further detail in earlier chapters of this document. As a result of these varying methods used for particulate measurement, especially in earlier studies, interpretation and extrapolation of health data for regulatory and air management purposes are difficult. The more inhalable smaller particles (<10 μm in size) have been implicated as more likely to penetrate and deposit themselves in the respiratory passages and lungs (see Chapters 10 and 11). In 1987, the US EPA replaced the previously used TSP standard by PM_{10}. Almost a decade has passed, and many studies have now been published that have measured PM_{10} directly and have attempted to associate these levels with various health effects. Additionally, several studies that measure fine particles, as $PM_{2.5}$ or PM_5, BS, sulphate, and/or particle strong acidity (PSA or H^+) have recently become available, and have been given special consideration in this review. It has been postulated that they may be more dangerous to health than TSP or PM_{10}, since their smaller size means that they are more able to reach the lower respiratory tract and lung (Seaton et al., 1995; Oberdoerster et al., 1995). In addition, some fine particles penetrate more readily than larger particles from ambient air into the indoor environment where most people spend most of their time, thus presenting increased opportunity for exposure (Chapter 6). The role of chemical speciation of particles has been investigated to a very limited extent. In several epidemiological studies, sulphate (SO_4^{2-}) has been used as the particle metric; a smaller number have investigated the role of acidity, as hydrogen ion (H^+). Some laboratory animal studies suggest that numbers of particles may be more important than particle mass, since ultrafine particles have a much greater surface area on which toxic substances such as metals or PAH can be adsorbed (Chapter 10). Very little epidemiological testing of this hypothesis has as yet taken place.

12.1.2 Study Types

Five basic health variables have been examined in these studies: mortality, hospital admissions/emergency department visits, respiratory health (symptoms, medication use, reduced activity days, elementary school absences), pulmonary function, and cancer (the latter in only a few studies, incidental to other endpoints). All of the epidemiological studies were observational in nature, that is, the investigator had no control over the exposure or treatment of the subjects in the study. These studies contrast with experimental studies (considered in Chapter 11), in which the investigator assigned the subjects to known exposures to particulate matter as well as other experimental conditions (e.g., temperature). It is generally easier to be certain of the cause-effect relationship from results of experimental studies than observational studies. One of the disadvantages with the observational study compared to the experimental study is that it is much more difficult to eliminate potential confounders. While some study designs allow for many of these potential confounders (for example cohort studies can take factors such as cigarette smoking and age into account on an individual basis), it is impossible on theoretical as well as practical grounds to account for all known and unknown potential confounding factors. This inability causes uncertainty with respect to the causal nature of an association between exposure to particulate matter and the observed health outcomes. This uncertainty requires that the data be examined especially carefully if causality is to be entertained, not only with respect to the conduct and results of each study, but with respect to interpretation of the overall findings from all of the studies, including relevant knowledge gained from experimental results.

Observational epidemiology studies fall into two broad categories: longitudinal studies where subjects or populations are followed over time, and cross-sectional studies where subjects are assessed for health outcomes at a particular time, at which time exposure is also ascertained (this can be present or past exposure). Cross-sectional studies usually compare individuals or population in several different locations, in order to be able to sample a wide range of exposures. True cross-sectional studies, in which several populations are compared with respect to health outcome and exposure, are considered to provide the weakest evidence for relationships between adverse health and exposure to particles (or other toxic agents), because differences (e.g., lifestyle factors, occupational exposures, age) between populations make the results highly subject to uncontrolled confounding and bias. Few of the newer studies investigating air pollution have been of this design, although some have retained certain elements of this. For example, the time-series six-city daily mortality study (Schwartz et al., 1996a) and two cohort mortality studies (Dockery et al., 1993; Pope et al., 1995a) both use community-based exposure estimates. The time-series study is not so subject to confounding by differences in populations since they are not correlated with changes in pollutant levels on a daily basis. The cohort mortality studies allow the analysis of the results incorporating most of the known risk factors on an individual basis. Both of these types thus provide stronger evidence for relationships than the true cross-sectional study.

Longitudinal studies are also of two main types: cohort studies and time-series studies. Some additional longitudinal designs have been carried out, such as analysis before, during and after pollution episodes. In a cohort study, subjects are identified and their varied exposures to the agent under investigation is determined, after which they are followed over a specified time to observe the effect of the exposure on health outcomes. Several recent studies have been of this type, in which thousands or hundreds of thousands of individuals in a number of communities have been recruited, categorized with respect to all of the major known potential confounding demographic or lifestyle factors, and their mortality followed over 6 to 16 years with respect to their community exposures to air pollutants. Cohort studies are generally considered to have the strongest design of all the observational studies, since they are able to include controls on an individual basis for most of the important known confounding factors, and to make inferences regarding groups made up of individuals with a given set of measured risk factors.

Most of the recent studies on air pollution have been the second type of longitudinal design, the time-series study. In this type, a time-series composed of changes in daily (or a few days') health indicators such as mortality or hospitalizations (usually data from very large administrative databanks) are followed for periods which have varied between 1 and 19 years, with respect to another time-series of daily variations in pollutant levels. The association of the two series is evaluated after control of other factors (weather, seasons, year-to-year trends, day-to-day and holiday variations, disease epidemics) which also vary with time and which could therefore bias the interpretation of the association. It has been pointed out (Lipfert 1994; Kinney et al., 1995) that the time-series study is less likely than the true cross-sectional study to give results that are potentially confounded due to population differences, since the study population in the time-series analysis is the same over time, and acts as its own "control". It is subject to confounding due to time-based differences, but these are easier to take into account in the analysis than population risk factors such as smoking status. A major advantage of the time-series study is that it usually provides many more units of observation (typically 1000 days) than cross-sectional studies (2-150 communities), and thus the power of the time-series study to detect effects of low magnitude is usually greater than the power of the cross-sectional study.

All of the observational studies available to date on air pollution are either "ecological" in nature or have some ecological elements. An ecological study is one in which the health outcome is measured for each exposure group and there are no data available on risk factors for individuals in the population (e.g., previous history of disease, diet, smoking, age, race), or on individual exposures (e.g., measurement from a few monitors for the entire city suffice for all members of the population). A direct link between exposure of the individual to the air pollutant and the resulting health outcome is lacking. Therefore no judgements regarding causality or risks to individuals can be made from ecologic studies (the ecological fallacy consists in making inferences from the population to the individual level). Some commentators (primarily from industry) on the research findings from air pollution studies have tried to dismiss the findings from time-series studies on the basis that they were "only ecologic studies". However, from a public health perspective we are as interested in and concerned about the air quality of the community as we are of the air quality of individual members of the community. Guidelines, objectives and standards are developed for community exposures, on the basis of information from population responses resulting from prior community exposures. Ecological studies can provide important evidence regarding causal relationships between community levels of air pollution and community health.

12.1.3 Time-Series Studies

Most of the recent epidemiological studies considering acute and short-term effects of air pollution on human health have been time-series studies in which the timing of an adverse health event (for example, hospitalizations) is studied in relation to short-term time trends in air pollution within a defined geographic area. Since these form a highly specialized type of epidemiological study, a discussion of their methods, strengths and limitations is included here.

Observational studies try to find relationships between population or community health responses and some characteristic of the environment, in this case air pollution from particulate matter. These relationships are often of low magnitude from a statistical point of view, the relative risk being less than 1.5 in many cases. Moreover, there is intrinsically a high degree of variability in mortality, hospitalizations, and other adverse effects that is unrelated to air pollution. A rigorous statistical analysis is therefore necessary in order to screen out adverse health effects due to non-pollutant-related factors and to have some hope of distinguishing the air pollution "signal" from the background "noise".

Daily mortality and morbidity (hospitalizations, emergency department visits, etc.) are usually highly cyclic, and undergo strong seasonal fluctuations, with events such as hospitalizations highest in winter and lowest in summer in North America. Ambient particulate matter concentrations can also be seasonal, and their highest levels may or may not coincide with the cyclic fluctuations in health endpoint, depending on location and source of the pollutant. Such seasonal trends could confound the results, and they require some means of adjustment in order to study the question of whether there is any independent association or effect of the pollutant on these health endpoints.

A common method of removing cyclic variations in the data is prefiltering through the use of a multiday (e.g., 15 d or weighted 19 d) moving average, by subtracting from each data point prior to analysis. This is called a "high pass" filter because it allows high frequency fluctuations (daily or weekly) to remain while removing the longer cycles from the data. Regressions with sine and cosine functions of various periodicities are also relatively effective in removing seasonal variations in data. Seasonal dummy variables have been used, but they cannot control for within-season variability. Restriction of the analysis to one season or to a part of a season (1 or 2 months) is another method to overcome seasonal variability (Kinney et al., 1995), but requires large data sets to overcome the loss of part of the data. A fourth method is the use of a nonparametric smoothing technique, such as the STL (Seasonal and Trend decomposition using LOESS, or the locally optimal estimating and smoothing scatter plots) method for estimation (Schwartz, 1996; Cakmak et al., 1996).

Temperature, both high and low, is also associated with adverse health effects, including mortality. Moreover, the strength of the association can be as great or greater than the strength of the particulate-mortality association. For example, a greater part of the variability in mortality was shown to be explained by weather and temperature than by air pollution (Kalkstein and Davis 1989). Temperature can be controlled for in a variety of ways; for example, the non-linear relationship of temperature with disease mortality can be accommodated by separate variables for high and low temperatures, quadratic variables, or by nonparametric smoothing techniques. An additional method of analysis included temperature as one of a number of aspects of weather affecting air masses, grouped as synoptic categories (Kalkstein 1991).

Other weather-related variables include lag time, that is, the time difference between a day with a given pollutant concentration and the day on which the health effect is manifested. Most authors have investigated lag times and included them in the analysis as the data warranted. Autocorrelation is also possible (pollution on one day may not be independent of pollution the next day, or a health outcome on one day may not be independent of health on the following days), and statistical methods are available to test for and overcome this (Schwartz et al., 1996b).

The detrended data are then examined using correlation analysis, regression analysis, or a combination. Regression analysis has commonly been used in recent papers, preceded by correlation analysis in order to give some direction to the regression analysis. It is not obvious at this time which regression model best fits the data, and no one model has been selected as preferential. As stated by Thurston and Kinney (1995), "in time-series analysis, no model is perfect, but some models are more wrong than others". Poisson regression has been extensively used in the mortality data because mortality is a rare event, following a Poisson distribution. Log normal distributions, and Ordinary Least Squares (OLS) regressions are also common for hospitalization data when large numbers of events are recorded. The results frequently do not appear to be very sensitive to which of these is used (Kinney et al., 1995; Lipfert 1994). Many of the most recent papers using administrative data bases have made use of these more sophisticated statistical techniques, to good effect.

12.1.4 Multiple Pollutants

Airborne pollution alway occurs as a mixture of agents, of which particulate matter is only one. Perhaps the most difficult issue to deal with in drawing any conclusions about the relationship of particulate matter to adverse health outcomes is the presence of other covariate air pollutants and the methods used to disentangle the influence of particulate matter from these other gaseous air pollutants.

Particulate matter, especially in the smaller size fraction, is a primary air pollutant, along with SO_2, NO_x, and CO; these are produced directly from combustion processes (see Chapters 2-4). Sources in North and South America and in western Europe are mostly vehicular traffic, industrial activity and some coal- or oil-fired power generation. Moreover, SO_2 and NO_2 can be transformed into secondary particulates by photochemical processes (into acid or neutral sulphates and nitrates for example) which become part of the fine particle fraction. These four air pollutants are often, (but not always) highly correlated with each other. If variables that are not independent, (i.e., are moderately to highly correlated) are entered into a multipollutant model as independent variables, the basic assumptions of the model regarding independence will be violated and the results will be uncertain and indeed, could be altogether invalid. Therefore, on the basis of common sources and high correlations between them, it would not seem reasonable *a priori* to treat them together in a multipollutant model and expect to obtain accurate estimates of their independent effects on health outcomes. Ozone has a much lower correlation with particulate matter and the other primary pollutants (with the possible exception of NO_2), since it is a secondary air pollutant formed by photo oxidation reactions with NO_x and volatile organic compounds in the atmosphere. Therefore there is some *a priori* reason to expect that models including both ozone and PM would give reasonable estimates of the independent effects of each.

Some investigators (Lipfert 1994; Moolkavgar et al., 1996; Moolkavgar and Luebeck, 1996) have been concerned that the association which has been ascribed to particles is actually reflecting the association of one or more of these other air pollutants that may not have been considered in the analysis or even measured in the study location. These and other investigators have preferred to include several or multiple pollutants together in multipollutant models, despite the attendant problems of collinearity, in order to allay the suspicion that something has been overlooked.

Other groups of investigators have examined each pollutant alone in models in which the other major non-pollutant variables (weather, season, day-to-day effects) have been accounted for (Dockery et al., 1992; Pope et al., 1992; Schwartz 1993; Pope et al., 1995b; Schwartz et al., 1996b). They have preferred to rely on the association noted in various locations with differing climates, weather patterns, sources and mixtures of air pollutants. However, univariate analyses are considered likely to give biased estimates of effects because of the possibility of more than one causal agent, and because of the high correlations between pollutants.

A modification of this approach has been extension of the analyses to include bivariate regressions with all pollutants that showed associations with adverse health outcomes in the univariate models, accompanied by correlation analyses showing that the correlations between the two are not excessively high (correlations greater than 0.5 would cast doubt on the validity of the results). Pollutants which were not associated in the original univariate analysis were considered unlikely to show associations in pairwise models. This approach is considered to be useful in helping to disentangle the effects of one pollutant from another and in establishing their independent effects within the same locality.

One problem that could occur is modification of the effects of one pollutant by the co-occurring pollutant included in the model. Interaction terms could be developed for the model (Schwartz et al., 1996b), or the effect of high and low levels of one pollutant on the other could be separately analyzed as was done for several of The Air Pollution and Health: a European Approach (APHEA) studies in Europe (Touloumi et al., 1996; Ballester at al., 1996; Sunyer et al., 1996). Another more intractable problem is that of the effect of differential measurement error on the outcome when several pollutants are included together. In all studies, air pollutants have been measured at area monitors, sometimes at only one for an entire city or even larger area (e.g., eastern Tennessee – Dockery et al., 1992). The exposure of individuals in the population will be different than the value give by the ambient area monitor, and will depend on factors such as where they live with respect to the monitor, how evenly spread the pollutant is over the area, whether it has local sources, how much time the individual spends indoors and outdoors, what activities they take part in, and how well the pollutant infiltrates indoors (see Chapter 6). Moreover, the error will be different for each of the measured pollutants that were analyzed since they react differently in the air mass, they may have different local sources, or they may penetrate buildings more or less readily than other pollutants. Lipfert and Wyzga (1995b) have shown that this differential measurement error can bias the interpretation of bivariate or multipollutant analyses, because the pollutant with the least measurement error will have the tightest confidence limits and the effect estimate for the pollutant with the larger measurement error may include zero. This type of error is difficult to recognize or to quantify if suspected, but the possibility should be considered when evaluating the results of bivariate or multipollutant analyses.

While careful analysis and consideration of the results from bivariate analysis in each location are useful in establishing which of several air pollutants have an independent effect on health outcomes in that location, they do not tell us whether particulate matter is really the causal agent, or is acting instead as the best surrogate of the pollutants examined in that location for the air pollution effect. For the overall evaluation of the role of particulate matter versus other air pollutants in exacerbation of adverse health effects, reliance has been placed on the results from a large number of locations in three continents: locations differing in pollutant levels, mixtures of co-occurring air pollutants, climate, and sources. It would be unlikely that co-pollutants could confound the particulate matter-adverse health association in all of these disparate settings.

12.2 ACUTE EFFECTS – MORTALITY

A number of time-series studies, which have used more advanced statistical methods than studies prior to the late 1980s to investigate the association of measures of mortality with increases in daily or several days' concentrations of particulate matter (PM), have been completed in the past decade. The results of these studies have raised questions about the safety of current air quality standards/objectives in both Canada and the United States.

Various measures of particulate matter were used, including total suspended particulates (TSP), $PM_{10/15}$, fine particles or $PM_{2.5}$, sulphates (SO_4^{2-}), and particle strong acidity (PSA or H^+). At the risk of losing valuable information on the nature of the particulate matter-adverse health associations which are provided by studies using TSP as the particle metric, this review on acute mortality effects has concentrated on studies that used inhalable particles, that is, PM_{10} and/or smaller size fractions including $PM_{2.5}$,SO_4^{2-} and H^+. Since the primary purpose of this review was to provide a scientific basis for selection of the National Ambient Air Quality Objectives for PM_{10} and $PM_{2.5}$, and since a relatively large number of studies were available that had used PM_{10}, this was felt to be justified. Particular attention has been given to studies that included both PM_{10} and one or more other measures of fine particles, with the objective of elucidating which of the PM components or properties might be the best indicator for the association of air pollution with adverse health. Studies published by December 1996 from locations in North America, South America and Europe, analyzed by regression techniques that tried to take account of many of the potential confounding factors noted previously, were included. Nineteen studies have been summarized in Table 12.1.

12.2.1 Acute Mortality Studies and PM_{10}

Daily mortality and PM_{10} pollution were studied in the Utah Valley between April 1985 and December 1989 (Pope et al., 1992). During winter temperature inversions, air pollutants were trapped near the valley floor, a situation that often resulted in high concentrations of PM_{10}, primarily due to emissions from a steel mill. The steel mill contributed approximately 82-92% of the PM_{10} emissions in the area. Monitors in Lindon, Provo and Orem collected PM_{10} data along with records of SO_2, NO_2, O_3 and aerosol strong acidity levels, all of which were found to be very low by comparison to other locations. The concurrent-day mean PM_{10} level was calculated to be 47 μg/m^3 (range: 1-365 μg/m^3).

Poisson regression with the generalized estimating equation of Liang and Zager was used to estimate mortality associations, correcting for autocorrelations. Adjustments were made for seasonal and yearly time trends, temperature, humidity, and if necessary, for temperature-PM_{10} interactions. A strong statistically significant relationship between PM_{10} pollution and mortality was found. Mortality averaged approximately 4-5% higher for each 50 μg/m^3 increase in concurrent-day PM_{10} pollution. On days when PM_{10} levels exceeded 100 μg/m^3, mortality was approximately 11% higher than on days when PM_{10} was less than 50 μg/m^3. Circumstances also allowed for a comparison of mortality when the mill was open or closed. Actual deaths per day were about 40% higher than expected when the mill was open. The strongest PM_{10}-mortality association was with the 5 d moving average (lag day 0-5) of PM_{10}. An increase in PM_{10} levels of 100 μg/m^3 (average lag 0-5) was associated with a 16% increase in total mortality (t=4.78). The association was greatest for respiratory deaths, at 43%, followed by cardiovascular deaths at 20%. Dose-response information based on division into quintiles indicated that the relative risk increased monotonically with increasing PM_{10}. The PM_{10} association was robust to a number of alternative model specifications to account for seasonality and weather (Pope et al., 1992). Since the gaseous pollutants were very low in this location, the study has been considered to provide strong evidence for the specific contribution of particulate matter to mortality (Pope et al., 1995b; US EPA 1996). It was noted by Thurston (1996) that CO, which is a risk factor in cardiovascular disease (CV), was not considered in the analysis, although it exceeded the US ambient standards on a number of occasions and thus could have acted partly as a covariate.

In a re-analysis and extension of the Pope et al. (1992) data, Lyon et al. (1995) extended the analysis by three years to 1992, and confirmed the previous findings by Pope that PM_{10} concentrations above 50 μg/m^3 increased daily mortality by about 4%, However, Lyon et al. maintained that additional analysis of the data by individual year, season, age of decedents, or location at death revealed inconsistencies in the association between PM_{10} air pollution and daily mortality, such that any question of causality was ruled out.

Table 12.1 Summary of Daily Time-Series Mortality Studies

LOCATION (population) (duration, dates) AUTHORS	**PARTICLE METRIC, µg/m³ (MEAN; RANGE)**	**RELATIVE RISK @ 100 µg/m³ increase. (95% CI) (Lag, d)**	**OTHER POLLUTANTS CONSIDERED (µg/m³ or ppb)**	**RELATIVE RISK @ 100 µg/m³ (95% CI) (Lag, d)**	**CO-REGRESSION, INTERACTIONS**	**COMMENTS, OTHER**
Utah Co. US (<500,000) (3.7 y, 1985-89) Pope et al., 1992	PM_{10} (47; 1-365)	1.16 (t=4.8) (all-c.) 5-d moving av.) 1.43 (respiratory) 1.20 (CV)	H^+ (below d.l.) SO_2 (negli.) NO_2 (low) O_3 (low in winter when PM_{10} high	not regressed	not considered to be necessary to correlate or co-regress other air pollutants because of low levels – Analyzed in later work (EPA 1996) O_3 negatively correl. with PM, strengthening PM effect PM_{10} RR 1.38 (0.98-1.94) +O_3	– Strong assoc. between PM_{10} and mortality was robust to changes in model specs., – coherent with morbidity results, same area. – Stat. analysis was careful. – Considered strong support for PM effect – Major criticism is lack of quantitative assessment of other air pollutants. CO, known to be above std., was not tested.
Utah Col US (<500,000 (7.7 y, 1985-92) Lyon et al., 1995	PM_{10} (as above)	1.04 (all-cause) (5d (0.98-1.10) (5d av) 1.13 (CV) (1.04-1.24 1.03 (resp) (0.86-1.3 1.08 (wntr; 0.98-1.2 1.13 (spr., 0.94-1.36 (summer., fall n.s.) (Indiv. years. n.s.)		Not considered		Despite dichotomization of PM_{10} data and consequent loss of data points, PM_{10} RR were signif. or marg. sign. in several cases. – did not succeed in proving that Pope et al. (1992) results were inconsistent
Salt Lake Co, Utah (4.5 y, 1985-1990) Styer et al., 1995	PM_{10} (48; 9-194)	n.s. for year, season (elderly, >65 y)	none considered		wide variety of weather and seasonal corrections included, but no other air pollutants	Complex statistical treatment makes interpretation of negative results difficult
Birmingham US (900,000) (3.4 y, Aug 85-Dec88) Schwartz 1993	PM_{10} (47.9; 21-80 (10th, 90th; 163 max)	1.11 (lag 1,2,3 mean) (1.02-1.20)	none considered			– PM_{10} was assoc. with mortality, warm city – Statistical analysis good; sensitivity analysis done; same results found with different models, different methods of control for weather, temp., epidemics, etc. – Major lack is not considering any other air pollutants which could act as covariates

Table 12.1 Summary of Daily Time-Series Mortality Studies *(continued)*

LOCATION (population) (duration, dates) AUTHORS	PARTICLE METRIC, µg/m³ (MEAN; RANGE)	RELATIVE RISK @ 100 µg/m³ increase. (95% CI) (Lag, d)	OTHER POLLUTANTS CONSIDERED (µg/m³ or ppb)	RELATIVE RISK @ 100 µg/m³ (95% CI) (Lag, d)	CO-REGRESSION, INTERACTIONS	COMMENTS, OTHER
Los Angeles Co. US (6 Million) (6 y, 1985-90) (only 364 d; 6-d monitoring) Kinney et al., 1995	PM_{10} (58±23; 15-177)	1.05 (Lag 0) (1.00-1.11) (RR = 1.03-1.09 for 14 combinations of temporal cycles, model, and weather)	O_3 (Lag 1) CO	O_3 only 1.02 (1.00-1.05) O_3 +PM 1.00 (0.94-1.06) CO only 1.07 (1.01-1.13) CO + PM 1.05 (0.99-1.12)	PM_{10} remained sign. with O_3 incl. PM RR =1.05 (1.00-1.11) with O_3 – O_3 n.s. (But only 364 d of both) CO found in prev. anal to be sign. PM RR 1.04 (0.98-1.09 with CO) – PM assoc weaker, marg. with CO – CO also weaker, marg. with PM	PM_{10} association was robust to different methods of control for cyclic var., weather, type of stat. anal, and other pollutants.RR = 1.02-1.09 for 17 different methods. – O_3 was not indep.assoc. with mortality – CO was independently assoc. But PM remained also indep. assoc. – SO_2 not considered, but low in LA – NO_2 not considered; known to be high
Chicago (Cook Co.) US (5.3 million) (6 y, 1985-90) Ito & Thurston, 1996	PM_{10} 40.7 ± 19.1 (20-65; 10th and 90th centiles (overestim. white suburbs- city monit.)	(Adults, age 15+) 1.05 (Av., L0+1) (1.03-1.08) 1.14 (1.04-1.25) resp 1.12 (1.06-1.18) canc 1.03 (0.98-1.07) circ	O_3 (38 ppb) SO_2 (24.5 ppb) CO (2.05 ppm)	1.10 (1.06-1.15) (lag 1day) 1.10 (0.95-1.3) resp 1.08 (0.99-1.18) canc 1.14 (1.05-1.19) circ. n.s. n.s.	Correlation coefficients: r= $PM_{10} - O_3$: 0.37 (moderate) PM_{10} – temp: 0.17 (low) O_3 – temp: 0.33 (moderate) temp – mortality 0.4-0.5 (mod-high) Coregression, PM-O_3 (+temp, etc) PM_{10} 1.04 (1.01-1.07) (L av 0+1) O_3 1.07 (1.01-1.12) (L av 0+1) (both lower than single regression but both significant)	– Provides good evidence that PM_{10} is assoc with mortality. Assoc robust to other pollutants, esp. O_3, which was independently assoc. with mortality. – PM showed cause specificity -elev RR for respiratory, cancer mortality but not CV. – Temperature also had incr. RR and was carefully analysed in several ways. – Authors thought some residual confounding of O_3 by hot temp. remained – Race and gender-specific RRs were developed (elev for blacks, females (B+w)) – Good analysis. Didn't look at NO_2.
Chicago (Cook Co) US (5.3 million) (6 Y, 1985-90) Styer et al., 1995	PM_{10} 38 (median) (3-365) (28-51 IQR or interquartile range)	(Elderly, age 65+) 1.054 ± 0.020 (yr) (n.s. in wint, summer) 1.088±0.03 (spring) 1.138±0.04 (F, 3 tms) 1.158±0.05 (F, 5 tms) (all age mortality.) 1.080±0.04 (Fall, 5) terms included in reg	none		no consideration of interactions with other air pollutants. Complex modelling of temperature, season, year, humidity, day of week, lag temp, tag humidity, lag d-of week. Different terms for each season included in Poisson regression	– Did not provide good evidence that PM_{10} was associated with mortality in the elderly – RR signif. raised in fall and spring but PM_{10} was highest in late spring and summer – Reason for difference between Styer results and Ito & Thurston results may be due to treatment by season. All year mortality RR was similar. I & T (96) didn't analyze by season.

Table 12.1 Summary of Daily Time-Series Mortality Studies *(continued)*

LOCATION (population) (duration, dates) AUTHORS	PARTICLE METRIC, µg/m³ (MEAN; RANGE)	RELATIVE RISK @ 100 µg/m³ increase. (95% CI) (Lag, d)	OTHER POLLUTANTS CONSIDERED (µg/m³ or ppb)	RELATIVE RISK @ 100 µg/m³ (95% CI) (Lag, d)	CO-REGRESSION, INTERACTIONS	COMMENTS, OTHER
Santiago, Chile (4.4 million) (790 d, 1989-91) (43,450 deaths) Ostro et al., 1996	PM_{10} (115; 32-367) winter mean 141 summer mean 76 (large proportion SO_4^{2-}, fine PM, from diesel)	(Poisson, full correct) 1.07 (1.03-1.10) Tot. 1.13 (1.08-1.23) resp 1.08 (1.03-1.12) cvd 1.10 (1.06-1.12) male 1.05 (1.00-1.09) fem. 1.10 (1.06-1.12) 65+ (OLS, min. Temp, tot) 1.10 (1.08-1.1.) (for mean increase of 115) (OLS, no corr, total mortality) @ mean inc 1.16 (1.14-1.18)	O_3 (1 h max) (53 ppb, 11-264) RR for yr. N.s. (OLS or Poisson) summer-1.04 (1.00-1.07) (Poisson) 1.02 (1.00-1.09) (OLS) NO2 (1 h max) (56 ppb, 10-258) RR 1.02 (1.01-1.04) OLS. N.s. Poi SO2 (1 h max) (60 ppb, 4-368)	n.s. for full yr (OLS or Ps) 1.04(Ps), 1.07 (OLS)-su 0.99 (y – Ps) 1.02 (y-OLS 1.04 (1.00-1.07) summer 1.01-1.02 (OLS, y, su, Poisson, yr., (all sign.))	correlation coefficients r = PM-O_3 -0.23; PM-SO_2 0.64 (PM-O_3 0.34-summ) PM-NO_2 0.73 Pairwise regr., OLS- y + su., Poi-y: PM_{10}: 1.04, 1.07, 1.04 (+O_3, NO_2, SO_2) 1.06 (0.97-1.11 – sum.,+ O_3) O_3+ PM: 0.97-0.99 (OLS, Poi, yr) 1.02 (1.00-1.05) (OLS, su 1.04 (1.02-1.09) (Poi, su) NO_2+PM: n.s. yr. or su (0.97-1.03)	– Good evidence that PM_{10} is assoc. With mortality. Robust to diff. methods of analysis. Extensive sensitivity anal. Resp. RR 1.15, CV RR 1.09 (cf total 1.08) Assoc. unchanged or stronger when regressed with NO_2, SO_2, for year or summer. PM_{10} weaker in summer with O_3 – O_3 independently assoc. In summer (RR 1.02-1.04) – comprehensive analysis, careful, except didn't consider CO
Sao Paulo Brazil (16 million) 1 y, May '90-Apr 91 Saldiva et al., 1995 Saldiva et al., 1994 (same as above)	PM_{10} (82.4 ± 38.8) as above	1.082 ± 0.019 (elderly ≥ 65 y) n.s. (children, respiratory mortality)	CO SO_2 NO_2 O_3 CO, SO_2, O_3 NO_2	1.13 ± 0.04 Significant Significant n.s. n.s., all 3 1.24 (1.13-1.34)	All pollutants were co-regressed – PM_{10} RR = 1.083 ± 0.033 in full model (same as alone) – CO, SO_2, NO_2 n.s. in full model correl. coeff. mod-high, 0.55 -0.68 NO_2 mod. highly correl. to others Without NO_2, no consistent relationships evident	– PM_{10} was the only pollutant which remained significant in the full coregr. – no seasonal analysis was done for O_3, which could explain neg. results. – PM_{10} had no effect – NO_2 was only pollutant related to respiratory mortality in children
Lyon, France (410,000) (6 y, 1985-90) Zmirou et al., 1996	PM_{13} 38; 5-98) (by radiometry, said to be equiv. to PM_{10})	n.s. (1.04- all-cause) 1.08 (respiratory) (1.00-1.18) 1.08 (CV) (0.99-1.10) (marg.)	SO_2 NO_2 O_3 (mean 1 h max. <20 all yr)	1.44 (resp) (1.1-1.8) 2.08 (CV) (1.4-3.9) n.s. n.s.	– PM_{13} and SO_2 coregressed – SO_2 sign. with PM_{13} but strength reduced; SO_2 RR=1.27 (PM_{13}<60) SO_2 RR = 1.19 (PM_{13}>60)	PM_{13} was not assoc. with all-cause mort. Weak association with respiratory, CV mortality – SO_2 hadvery strong assoc., slightly. reduced when PM in model – no effect for O_3; central monitor affected by NO_2 scavenging. – No CO (confounder in CV results?)

Table 12.1 Summary of Daily Time-Series Mortality Studies *(continued)*

LOCATION (population) (duration, dates) AUTHORS	**PARTICLE METRIC, µg/m³ (MEAN; RANGE)**	**RELATIVE RISK @ 100 µg/m³ increase. (95% CI) (Lag, d)**	**OTHER POLLUTANTS CONSIDERED (µg/m³ or ppb)**	**RELATIVE RISK @ 100 µg/m³ (95% CI) (Lag, d)**	**CO-REGRESSION, INTERACTIONS**	**COMMENTS, OTHER**
St. Louis, Mo., US (2.3 million) 1 y, Sept 1985-Aug 1986) Kingston-Harriman, Tenn., USA (640,000) (1 y, as above) Dockery et al. 1992	PM_{10} 27.6 (max 97) $PM_{2.5}$ 17.7 (max 75) $PM_{10\text{-}2.5}$ SO_4^{2-} 8.0 (max 38) H^+ 9.7 nmol/m³ PM_{10} 30 (max 67) $PM_{2.5}$ 21 (max 58) $PM_{10\text{-}2.5}$ SO_4^{2-} 8.7 (max 27) PSA (as H^+) nmol/m³	1.16 (L 1 d) 1.19 (p=0.075) 1.28 (p=0.056) weak pos., n.s. weak pos., n.s. 1.17 (n.s. P=0.28) n.s. n.s. n.s. n.s.	SO_2 (24 h) NO_2 (24 h) O_3 (24 h) as above	n.s. n.s. n.s. Similar to St. L; none of gaseous pollutants significant (reanalysis in EPA 1996: PM_{10} RR including O_3) St L: 1.12 (0.99-1.30) KH: 1.17 (0.97-1.52)	– pollutants regressed singly, except fine and coarse particle ($PM_{2.5}$ and $PM_{10\text{-}2.5}$) considered simultaneously – PM_{10}/SO_4^{2-} correl. coeff. = 0.52 – PM_{10}/H^+ correl. coeff. = 0.76	– PM_{10} was only pollutant assoc. with mortality in St. Louis. – $PM_{2.5}$ and CP marg. signif., about equal – SO_4^{2-} and H^+ weakly pos., but n.s. – Order was $PM_{10}>PM_{2.5}=CP>SO_4^{2-}>H^+$ Assoc. not stat. sign. in E Tenn. because of small pop., short time period, exposure missclass. – Effect of gaseous poll. not tested – reason for O_3 n.s. was 24 h averaging
6-CITY study, US St. Louis (2.3 M) E. Tenn (0.6 M) Boston, (2.3 M) Steubenville (0.2 M) Topeka (0.15 M) Portage (0.3 M) (5.9 M; 8 y, '79-87) Schwartz et al., 1996a	PM_{10} 25.0 (8.0-67.8) CP 9.0 (1.0-30.1) $PM_{2.5}$ 14.7 (4.3-431) SO_4^{2-} 5.8 (1.5-22.3) H^+ 10 (0-67) (nmol/m³) (medians, 5th +95th centiles)	1.08 (1.05-1.10 (2 d)) 1.04 (0.90-1.10 (2 d)) 1.15 (1.10-1.19 (2 d)) 1.22 (1.13-1.32) n.s. (t=0.76) indiv. city – $PM_{2.5}$: Bo 1.22 (1.15-1.29) ET 1.14 (1.02-1.26) SL 1.10 (1.04-1.17) St 1.10 (0.90-1.21) Po 1.12 (0.97-1.28) To 1.08 (0.80-1.36)	none (data for SO_2, NO_2, O_3 available)	not calculated	– purposely did not examine interactions between gases and PM – When CP and $PM_{2.5}$ co-regressed, PM RR was unaffected but CP RR was 0. Correlation co-efficents were low, 0.23-0.43, in 5 of 6 cities, but 0.69 in Steubenville, the city in which CP RR was sign. and $PM_{2.5}$ RR was not.	– $PM_{2.5}$ was particle most strongly assoc. with total mortality, also CV, COPD mortality (RR increased to 1.21, 1.33) – CP not assoc. in 5 of 6 cities – CP ruled out for several reasons: weak n.s. assoc.; low correl coeff; poor model fit with $PM_{2.5}$; CM RR prop to PM/CP ratio; low PM_{10} RR in 1 city with high CP – SO_4^{2-} also assoc., but not as strongly, and indep. of $PM_{2.5}$ assoc. – H^+ not assoc. (fewer measurements for H^+, but N=1600+, so enough power) – Carefully conducted, powerful study

Table 12.1 Summary of Daily Time-Series Mortality Studies *(continued)*

LOCATION (population) (duration, dates) AUTHORS	PARTICLE METRIC, μg/m^3 (MEAN; RANGE)	RELATIVE RISK @ 100 μg/m^3 increase. (95% CI) (Lag, d)	OTHER POLLUTANTS CONSIDERED (μg/m^3 or ppb)	RELATIVE RISK @ 100 μg/m^3 (95% CI) (Lag, d)	CO-REGRESSION, INTERACTIONS	COMMENTS, OTHER
Philadelphia, US (million) (2 summers, 1992-3) Dockery et al., 1996b	PM_{10} (not given) $PM_{2.5}$ (not given) CP ($PM_{10-2.5}$) (not given) H^+	1.12 (p=0.06, marg) 1.20 (1.04-1.35) (L 1) n.s. (p=0.85) n.s. (p=0.57)	O_3 SO_2 CO	1.09 (sing., but CI n.g.) n.s. (p=0.78) n.s. (neg. assoc.)	no information on whether co-regression or correlation analysis carried out	$PM_{2.5}$ was most strongly and consistently assoc. with mortality – ozone was also independently assoc. with total and non-CV mort. – ozone was not consistent by season, the RR being lowest in summer. – small study, short duration, abstract only
Toronto, Canada (2.5 million) (19 y, 1972-90) Ozkaynak et al., 1995	TSP SO_4^{2-} CoH b-extiction coeff. PM_{10} (meas + est.)	1.07 (from est PM_{10}) (1.05-1.09) (From Thurston 1996)	O_3 SO_2 NO_2 CO	1.047	(abstract; preliminary paper) Thurston 1996 give addit. Info. PM_{10} RR = 1.05 (1.03-1.07) with other pollutants in regression.	– work not fully reported. – PM_{10} was estimated (site-specific) from TSP, monitored PM_{10}, CoH, SO_4^{2-} and Bext. – PM and ozone indep. assoc. with mortality
Amsterdam, NL (700,000) (7 y, 1986-92) Verhoeff et al., 1996	PM_{10} (38; max 163) BS (12; max 81)	1.06 (marg.) (0.99 - 1.14) (L 0d) 1.19 (1.02 - 1.38) (L 0d)	O_3 SO_2 CO	1.05 (1.0 -1.10) (L 2d) n.s. n.s.	PM_{10} RR lowered, n.s. with SO_2, O_3 PM_{10} RR raised, CO BS RR sign. with O_3, SO_2, CO O_3 marginally sign. with BS O_3 significant with PM_{10}	– Supports importance of BS, finer particle size, (but not coregr.with PM_{10}) – PM_{10} effect partly due to O_3, SO_2 – ozone had independent effects – No analysis for NO_2 (possible confounder)
Paris, France (6.1 million) (6 y, 1986-91) (Dab et al., 1996)	PM_{13} (51; 19 – 137) BS (32; 11 – 23)	1.17 (1.04 – 1.31) (L 1d) 1.07 (marg) (0.98 – 1.18) (L 1d)	O_3, 1 h max. O_3, 8 h NO_2 SO_2, 24 h SO_2, 1 h max	n.s. n.s. Sign., in summer (L 1d) n.s. 1.07 (1.015-1.16) (L 1d)	None tested	– Stronger association for PM_{13} than BS which may not include all fine particles – SO_2 may play a role in this location – lack of interaction testing means uncertain of strength of PM association – CO was not tested -no seas. anal. for O_3 – possible reason for lack of significance

Table 12.1 Summary of Daily Time-Series Mortality Studies *(continued)*

LOCATION (population) (duration, dates) AUTHORS	PARTICLE METRIC, µg/m³ (MEAN; RANGE)	RELATIVE RISK @ 100 µg/m³ increase. (95% CI) (Lag, d)	OTHER POLLUTANTS CONSIDERED (µg/m³ or ppb)	RELATIVE RISK @ 100 µg/m³ (95% CI) (Lag, d)	CO-REGRESSION, INTERACTIONS	COMMENTS, OTHER
Athens, Greece (<3 million) (5 y, 1987-91) Touloumi et al., 1996	BS (84; 9 -333) [PM_{10}] [79]	1.05 (1.03 – 1.08) (L 1d) (1.054) (1.033 – 1.087) [site-specific conversion from BS]	SO_2 CO	1.12 (1.07 – 1.16) (L 1d) 1.10 (@ 10 mg/m³) (1.05 – 1.15) (L 0d)	BS RR lowered with SO_2; RR = 1.03 when SO_2<100; RR = 1.02 (n.s.) when SO_2> 100 SO_2 RR incr. To 1.15 (BS>100) BS, CO highly corr. (R=0.79) CO n.s. with BS in previous report (Touloumi et al. 1994)	– BS effect was partly independent of SO_2 – SO_2 plays a more important role than BS, or is a better surrogate for air pollutants – No analysis for NO_2 or O_3 (other possible confounders)
London, UK (8 million) (6 y, 1987-92) Anderson et al., 1996	BS (14.6; 8 – 23)	1.11 (all-cause, year) (1.05 – 1.17) (L 1d) (also sign., summer + winter alone) n.s. – resp. mort. n.s. – CV mort.	O3 1 h max. 8 h av. NO_2 1 h max NO_2 24 h SO_2 24 h	1.08 (all-cause, y) (1.04 – 1.12) (L 0d) CV – 1.04 (year) resp. – 1.07 (year) 1.10 (all-c, summer) sign., CV, summer n.s. sign., all-c., summer	– BS RR (all-c., y.,wi.) increased with O_3. – O_3 RR unchanged with BS (all-c.-su., CV- su..; resp.- y., su., wi.) RR for BS and O_3 unchanged with NO_2 or SO_2	– Both BS and O_3 were independently associated with mortality – O_3 a more important predictor than BS – BS was not assoc. with either respiratory or CV mortality – inconsistency CO was not included in analysis
Barcelona, Spain (million) (7 y, 1985-91) Sunyer et al., 1996	BS (49.7 – winter) (35 – summer)	1.07 (all-cause, y) (1.03 – 1.11) 1.09 – respiratory, y 1.10 (marg.)- CV, y	SO_2 O_3 NO_2	1.13, 1.15, 1.24 (all-c, y; CV, y; resp, su) 1.05, 1.06 (y, all-c, CV) 1.06, 1.09 (su, all-c, CV) sign., all-c., CV, both year + summer	– no bivariate regressions done. – Correlation coefficient for BS-SO_2 was high (r=0.69) – Correlation coeff. for BS-O_3-NO_2 were rel. low, (r= 0.29-0.33)	– BS was assoc. with all-cause, CV and resp mortality in single poll. analysis – SO_2 was more strongly assoc. than BS but high correl. coef. precludes separation of effects for these two pollutants. – Authors sugg. BS not best surrogate for vehicle-related PM pollution – O_3 effect real, and independent of BS – No CO anal.; could be CV confounder)
Valencia, Spain (0.75 million) (3 y, 1991-3) Ballester et al., 1996	BS (68; 5-226) (77 – winter) (58 – summer)	1.09 (1.03 – 1.15) (All cause, year) 1.12 (1.03 – 1.22) (cardiovascular) <1.0 (respiratory)	SO_2	1.07 (0.99 – 1.15) (all cause, summer)	No bivariate regressions. SO_2 -BS correlation 0.24 (relatively low)	

Closer examination of their analyses does not support their case very well. Subdivision of the data by year, season, age, or cause of death probably contributed to lack of significance of many of the outcomes because of low numbers of cases. Analysis of the data by year showed that the risk was not significant for any individual year, or for the total, although the relative risk (RR) reached marginal significance for two of the years and the total. However, when the analysis was carried out using a similar method with continuous data for the same years as the Pope et al. study, the regression coefficients were similar in the two studies. Similarly, seasonal analysis, with the highest risk ratio said to be in spring, was also cited as calling into question the evidence for PM_{10}, which is highest in winter in this location. The data presented indicate that the RR for winter was 1.08 (95% CI 0.98-1.19), while the RR for spring, although higher at 1.13, was more variable and was not significant (95% CI 0.94-1.36).

The authors also cited the evidence that the greatest RR was for people over age 75 years who died in hospitals as further evidence that particulate matter could not have been associated with mortality. They reasoned that exposure to particulate matter would likely be less in hospitals than outside or at home, and therefore could not have been involved. This assumption regarding exposure is not necessarily true, since the PM_{10} in this location was of industrial origin and thus was probably largely in the fine fraction which penetrates buildings quite easily (Chapter 6). In addition, people could be expected to go to hospital for emergency treatment and/or admission, and to die in hospital later, a circumstance which is usually accounted for by allowing for one or more days of lag time (in the Lyon study 3 d and 5 d averaged lag times were used) between the pollutant peak concentration and measurements of mortality. A lack of consistency was also cited with respect to specific cause of death, since the Pope analysis showed respiratory deaths to be most elevated, followed by cardiovascular deaths (CVD), while the Lyon analysis found that the RR for respiratory deaths was not significant and the CVD RR was elevated (RR=1.13; 95% CI 1.04-1.24). As with other analyses in the Lyon et al. study, the results appeared to be affected by high variability introduced by low numbers of events due to splitting up of the available data.

A major difference in treatment of data between the Lyon and the Pope studies, which also could have accounted for a substantial part of the difference in results, was the dichotomization of PM_{10} data using 50 $\mu g/m^3$ as the cutoff, as carried out in the Lyon et al. study (the 5 d moving average was used in both studies), and the consequent loss of data compared to the analysis of continuous data carried out by Pope et al. (1992). Since the fourth quintile average PM_{10} values in Lyon et al. (1995) were below 50 $\mu g/m^3$ but still showed a substantial increase in relative risk, these responses would have been discounted (i.e., given a value of 0) and only the upper 20-25% of data used, thus reducing the significance of the PM_{10} response in most of the subsequent analyses.

The dose-response analysis of Lyon et al. (1995) looking at quintiles of PM_{10} and associated responses, indicated that the RR was not elevated above 1.0 for the first three quintiles, but was elevated at quintile four (mean PM_{10} 47 $\mu g/m^3$) with a RR of 1.046 (no CI given), i.e., that a threshold existed between 36 and 47 $\mu g/m^3$. In contrast, the Pope et al. (1992) study had shown a relatively smooth dose-response curve in a similar quintile analysis without any evidence of non-linearity in response between 17 and 84 $\mu g/m^3$, but for a smaller data set in the same location. While comparison of Lyon's PM_{10} data for the extra three years did not reveal any striking changes in PM_{10} levels from the previous three years analyzed in both studies, the quintile division breakdowns showed substantial anomalies between the two studies. The mean of the first four quintiles in Lyon et al. (1995) were 5 to 9 $\mu g/m^3$ lower that those in the Pope et al. (1992) study, while the mean for the fifth quintile was 15 $\mu g/m^3$ above the fifth quintile of Pope et al. (1992). These anomalies indicate that considerable caution should be taken in weighting the evidence from the Lyon et al. (1995) study in the overall evaluation.

An analysis of PM_{10} and mortality in the elderly was conducted by Styer et al. (1995) for Salt Lake County, Utah, which is the neighbouring area to that considered in the Pope et al. (1992) study. It has approximately the same mean (48 $\mu g/m^3$) and seasonal pattern for PM_{10} as Utah County. A Poisson distribution was assumed for small counts (average 7/d), and factors for year, season, within-season trends, seasonal interactions with PM_{10}, temperature, humidity and barometric pressure were added to the models. Three-day average PM_{10} values accounted for possible lagged effects. In the resulting analyses, PM_{10} was never a significant predictor of elderly mortality in Salt Lake Co., with or without the addition of barometric pressure which a supplementary semi-parametric analysis indicated was important in Salt Lake. No seasonal or within-season effects of PM_{10} on mortality were indicated. Since the authors in this study used the same general methods as Pope et al. (1992) and other investigators, i.e., testing of various models incorporating the effects of weather, day to day, and season by Poisson regression techniques, the methods used did not suggest any apparent reason to the reviewer for the negative results, other than the suspicion that the models used by Styer et al. (1995) overcorrected for interactions between PM and weather.

Schwartz (1993) studied the impact of air pollution on daily mortality in Birmingham, Alabama, between 1985 and 1988. Daily death counts from detailed mortality tapes of the National Center for Health Statistics were matched to 24 h average PM_{10} levels from all the population-oriented monitors within the city. The reported mean PM_{10} level was 47.9 $\mu g/m^3$, with a maximum of 163 $\mu g/m^3$. No specific sources of the pollution were mentioned. Concentrations were highest in summer and lowest in winter, the opposite of the trend in daily mortality. Significant associations were found between PM_{10} and daily mortality at concentrations below 150 $\mu g/m^3$, the 24 h air quality standard. The relative risk calculated for every 100 $\mu g/m^3$ increase in the mean of the three days' previous PM_{10} was 1.11, with a 95% confidence interval of 1.02-1.20. The RR was virtually unchanged when other regression models or alternative methods of controlling for temperature and seasonality were used. The results were not sensitive to high pollutant levels, and the response was evident down to the lowest level of PM_{10} (20 $\mu g/m^3$). The relative increase in mortality was higher for respiratory and cardiovascular deaths and lower for all other causes, suggesting a similar etiological factor for these two endpoints. This study carefully controlled for seasonal and meteorological variables, and carried out sensitivity analyses to ensure that the associations were not biased. The results of this study thus clearly support the association of air pollution, as represented here by PM_{10}, with increased mortality. However, since this analysis did not consider ozone, it cannot exclude the possiblilty that ozone, which is often not highly correlated with PM due to somewhat differing conditions for formation, is also independently associated with mortality in this community.

Kinney et al. (1995) conducted a study on mortality and daily variations in particulate matter in Los Angeles, California where the mean PM_{10} values were calculated to be 58 $\mu g/m^3$, with a maximum of 177 $\mu g/m^3$. The relative risk for a 100 $\mu g/m^3$ increase in PM_{10} was reported to be 1.05 (95% CI 1.01 to 1.10) when regressed alone. The relative risk and its significance was unchanged for PM_{10} when regressed with ozone, but ozone was no longer significant (RR=1.0). Substantial correlation was reported in this location between ozone and PM_{10}, which made it impossible to separate out any possible independent effects of ozone when they were both included together. The relative risk for PM_{10} dropped to 1.04 (95% CI 0.98 to 1.09) when regressed simultaneously with CO. The relative risk for CO also dropped slightly from 1.07 alone to 1.05 (95% CI 0.99 -1.12) with PM_{10}. The correlation between PM_{10} and CO was somewhat less than that for PM_{10} and ozone. This study showed that air pollution, as measured about equally by both PM_{10} and CO, was associated with mortality in Los Angeles.

An analysis of mortality data for the six years from 1985-1990 for Chicago and Cook Co, Illinois was presented by Ito and Thurston (1996). The mean PM_{10} was reported as 40.7 $\mu g/m^3$ (10th and 90th percentiles 17 and 65.6 $\mu g/m^3$). The relative risk for total non-accidental mortality for a 100 $\mu g/m^3$ increase in PM_{10} was 1.05 (95% CI 1.03-1.08) (single pollutant regression) and 1.04 (95% CI 1.01 to 1.07) for a two-pollutant model including ozone. Ozone was independently associated, but the RR for ozone was reduced more than PM_{10} when both were included. The authors also pointed out that the pattern of associations seen for ozone closely mirrored those for temperature, which was known to be correlated with ozone concentrations, suggesting that the effects noted for ozone could have been explained by temperature effects. Cause specificity was demonstrated with PM_{10}, with elevated relative risks for respiratory and cancer deaths, and marginally elevated RR for cardiovascular deaths, while ozone was not associated with an elevated relative risk for respiratory deaths as might have been expected. CO, SO_2, and the extinction coefficient b_{ext}, a measure highly correlated with fine particulate matter, were also considered but were not as good predictors of an air pollution effect on mortality as PM_{10}, which was the best predictor in this study.

Mortality in the elderly, age 65+ years, was examined by Styer et al. (1995) in a second analysis for Cook

Co., IL (Chicago). For the six years 1985-1990, the median PM_{10} concentration was 38 μg/m^3 (range 3-365 μg/m^3), and the interquartile range was 28-51 μg/m^3. An average of 117 nonaccidental deaths/day (all ages) and 83 deaths/day (age 65+) were recorded, and a total of over 18,000 deaths in the elderly were analyzed. A high degree of association of PM_{10} with meterology (correlation coefficient of PM_{10} with daily mean temperature = 0.52) and a lesser degree of association of PM_{10} with mortality was found. Various treatments for year, season and weather were examined in regression models, including terms for each year, cube of the day of year, polynomial terms for temperature lagged one and two days, humidity on day 0 and lagged two days, barometric pressure and a polynomial term for pressure lagged one and two days. These were examined in a semi-parametric Poisson regression model. The majority of analyses split data by three-month season, and also month-by-month, both of which would have the effect of reducing the number of underlying observations, thus reducing the power of the study to detect an effect.

The whole year estimate for PM_{10} (3 d average, lag 0-3 d) gave a relative risk of 1.055 (95% CI 1.01-1.10 for a 100 μg/m^3 increase) for persons aged 65 or more, which was similar to the RR estimated by Ito and Thurston (1996). However, the seasonal estimates were significant only in spring and fall, which did not coincide with the season of highest PM_{10} concentration (summer). The spring and fall results could not be explained by high pollen counts, which were available and were accounted for in the analysis. Since PM_{10} in the fall season showed the highest association with mortality, this season was analyzed in greater detail, using various model predictors for each category. The RR for total mortality (all ages), elderly mortality, males aged 65+, whites aged 65+, and cancer deaths were significantly elevated, while RR for females and blacks aged 65+, circulatory deaths and respiratory deaths were not. The authors considered that these results showed inconsistencies, probably due to the lack of ability of regression analysis to deal adequately with weather conditions which may have confounded the results. They concluded that the reported effects of particulate matter on mortality were unconfirmed (Styer et al., 1995). The possibility also exists, and the reviewers considered, that they have overcorrected for season and weather, especially in the analyses by season, race and age which were reduced in size, and therefore in power to detect an effect. This study is therefore also considered to give positive support for the association of air pollution, as measured by PM_{10}, with mortality.

A daily mortality study was carried out in Santiago, Chile during 790 days over a three year period from 1989-1991 (Ostro et al., 1996). 43,450 deaths were recorded over this period, with a mean of 55.0 deaths/day. Air pollution levels were much higher in this city than in comparably sized North American locations. The mean PM_{10} was reported to be 115.4 μg/m^3 , with a range of 35-500 μg/m^3 . The mean PM_{10} in the summer was 76.2 μg/m^3 and 141.4 μg/m^3 in the winter. A large proportion of the PM_{10} was stated to be sulphates and other fine fraction PM, from diesel sources (74%) and gasoline vehicles (6%). Analysis was by Poisson and normal (Ordinary Least Squares) regression. Based on an increase of 100 μg/m^3 in PM_{10}, the relative risk for total mortality was 1.07 (95% CI 1.03 to 1.10), and was 1.08 (95% CI 1.06 to 1.12) for an increase equal to the mean of 115 μg/m^3 PM_{10} (this was the value reported in the paper, based on Poisson models). The relative risk for respiratory mortality was higher than that for total mortality at 1.13 (95% CI 1.07 to 1.20), while cardiovascular mortality was similar to total mortality at a relative risk of 1.08 (95% CI 1.03 to 1.12) . Mortality was also elevated above the mean for the elderly, age $\geq$ 65 years, with a relative risk of 1.10 (95% CI 1.06 to 1.12) (all were adjusted for yearly, seasonal and temperature variables, and scaled down from increases equal to the mean of 115 μg/m^3 to increases per 100 μg/m^3, for easier comparability with results from other studies).

Ozone (lagged 1 day) was significantly and independently associated with mortality in summer (Poisson RR= 1.04; 95% CI 1.00 to 1.09 for a 100 ppb change), but not in winter or over the whole year. In OLS analysis of the summer-only data, the RR for PM_{10} alone was marginally significant (RR=1.07; 95% CI 0.99 to 1.11 for 115 μg/m^3), but the RR for both PM_{10} and ozone were slightly reduced when both were included together in bivariate analysis (PM_{10} RR=1.06; 95% CI 0.97 to 1.11, and ozone RR=1.02; 95% CI 0.97 to 1.07). SO_2 was marginally significant and NO_2 was not, in univariate analyses, but both were not significant when each was considered with PM_{10} in bivariate Poisson regressions. CO, one of the standard air pollutants, was not considered, although it was undoubtedly very high in this traffic-polluted city. The association of PM_{10} with mortality remained significant in a number of different regression models with and without other gaseous pollutants, and with extensive sensitivity analyses. This study provides good evidence that air pollution associated with high vehicle traffic, as indexed by PM_{10}, is robustly associated with mortality. Summertime ozone levels also appeared to have an independent association with mortality, along with PM_{10}.

Saldiva et al. (1994) studied children aged < 5 years old and respiratory mortality in Sao Paulo, Brazil. The mean PM_{10} was 82.4 ± 38.8 µg/m^3 (higher than comparable North American locations) and the mean nitrogen oxides (NO_x) was 127 ppb. The other criteria pollutants O_3, SO_2, and CO were also considered. This study included only one year of data from May 1990 to April 1991, the mean deaths being low at 3/d. PM_{10} was not associated with respiratory mortality in young children (slight negative correlation). NO_x was the only air pollutant with significant effects (odds ratio of 1.24, 95% CI 1.13 to 1.34 for a 100 ppb increase). Moreover, when NO_x was removed from the analysis, no other consistent relationships were observed with the remaining air pollutants. NO_x was moderately to highly correlated with the other pollutants ($r \geq 0.55$ for PM_{10} with NO_x), but not with temperature or humidity. This study indicated that air pollution, in this case best indexed by NO_x and not by any other pollutant, was associated with effects sufficient to induce mortality in children.

In a second study in Sao Paulo, Brazil, Saldiva et al. (1995), examined mortality in the elderly aged 65 years and older. Mean PM_{10} levels were as reported above. Mean daily O_3 was 12.5 ppb with a mean of 1 h daily maximum of 38.3 ± 29.7 ppb. All five of the usual criteria pollutants were included, but as mentioned above the data were available for only one year. The relative risk for total mortality was 1.082 ± 0.019 ($p \leq 0.01$) for a 100 µg/m^3 increase in PM_{10} when no other pollutants were included in the regression, and was unchanged at 1.083 ± 0.033 when all other criteria air pollutants were included together in the model. The dose response was linear, and showed no evidence of a threshold when plotted by quintile of concentration ($p < 0.0001$). CO was associated with mortality ($p \leq 0.05$) when considered alone in the regression, with a relative risk of 1.127 ± 4.13 for a 7.5 ppm increase, but was no longer associated with mortality when it was considered together with the other pollutants. SO_2 and NO_2 also were associated with somewhat increased relative risks when each was considered alone, but were not predictive of increased mortality when considered at the same time as PM_{10}. Correlation coefficients between PM_{10} and SO_2, NO_2 or CO were high (0.66, 0.68 and 0.55) which makes separation of their effects impossible. Neither O_3 metric gave increased relative risk estimates either alone or with PM_{10} . A seasonal analysis was conducted for PM_{10}, but not for ozone, although ozone has been shown in several analyses to be predictive of increases in adverse health effects in summer, but not in winter (Burnett et al., 1994, 1995; Ostro et al., 1996). In this location and study, bivariate analyses consistently attributed the air pollution-mortality association to PM_{10} , which was clearly the best index for the air pollutant mixture of those examined.

Daily mortality and air pollution were examined in Lyon, France for the six year period from 1985-1990, as part of the European APHEA collaborative study (Zmirou et al., 1996). The mean PM_{13} , which was measured by b-ray atomic absorption, considered here to be equivalent to PM_{10}, was 38 µg/m^3 (range 4.8-97.5 µg/m^3). Particles were lowest in spring and highest in winter. The effects of SO_2, NO_2, and O_3 on mortality were also examined. Meteorological and seasonal factors were considered in the model. The RR for a 100 µg/m^3 increase in PM_{13} was 1.08 (95% CI 1.00 to 1.18) for respiratory deaths and almost the same (RR=1.08; 95% CI 0.99-1.10) for cardiovascular deaths (CVD). Few events, 0.4 deaths/d and 2.0 deaths/d, were recorded for both respiratory and CVD, and this probably contributed to the lack of a robust association. PM_{13} was not significantly associated for all-cause mortality (RR = 1.04; 95% CI 0.94 to 1.10). In this location, SO_2 (daily mean) was the pollutant most clearly associated with respiratory and cardiovascular (CV) mortality; it remained significant when considered together with PM_{13} (data not shown in paper). When PM_{13} was "high", (>60 µg/m^3), the RR for SO_2 was higher (RR=1.27) than when PM_{13} was "low" (RR=1.19). The correlation between SO_2 and PM_{10} varied considerably over the seasons in different monitoring locations, between -0.08 and 0.76, which allowed for an assessment of their interaction. No association was observed between ozone (1 h maximum) and mortality, which the authors suggested could have been due to the central placement of the ozone monitors in central city locations subject to ozone scavenging by traffic-generated NO_2. Thus this study supported a role for SO_2 as the best metric for expressing the association of air pollution with all-cause, respiratory and cardiovascular mortality, and for PM_{13} as a somewhat less effective predictor for respiratory and CV mortality than SO_2, at least under the conditions prevailing in this city.

12.2.2 Acute Mortality Studies Including PM_{10}, $PM_{2.5}$, and Other Measures of Fine Particulate Matter

Dockery et al. (1992) examined daily deaths from annual mortality data kept by the National Center for Health Statistics for the one year period from September 1985 to August 1986 in St. Louis and the counties of eastern Tennessee surrounding Kingston/Harriman. Particulates were measured daily. A dichotomous sampler was used to separate the particulate matter into fine (<2.5 µm) and coarse (<10 µm) fractions. Mean PM_{10} levels were 27.6 and 30 µg/m^3, with maxima of 97 and 67 µg/m^3 for St. Louis and Tennessee, respectively. $PM_{2.5}$ levels were also reported, with mean levels of 17.7 and 21 µg/m^3 and maxima of 75 and 58 µg/m^3 for the St. Louis and Tennessee regions, respectively. Mean sulphate levels were relatively similar in the two communities, at 8.0 and 8.7 µg/m^3 , with maxima of 38 and 27 µg/m^3 for St. Louis and Tennessee respectively. Particle strong acidity, measured as H^+, was much higher in Tennessee than in St. Louis, with mean levels of 36.1 nmol/m^3 compared to 9.7 nmol/m^3 for St. Louis. Other pollutants considered included 24 h mean SO_2, NO_2 and O_3. Ozone levels were the same in both regions. Carbon monoxide, which always accompanies combustion processes, was not considered. Sources of pollution were not specified, but it was speculated that there exists a mix of vehicular, industrial and natural sources. The association between mortality and air pollution was examined using Poisson regression analysis separately for each pollutant, after adjusting for season and weather variables.

Similar increases in daily mortality were found to be associated with PM_{10} levels on the previous day in both St. Louis (16.2% for a 100 µg/m^3 increase in PM_{10} ; 95% CI 1.01 to 1.30%), and eastern Tennessee (17.4%), but the 95% CI included the null value in the latter location (Dockery et al., 1992). In St. Louis, the proportion of total deaths attributable to PM_{10} during the 1 year study period was 4.2%. Recorded levels of PM_{10} were rarely in violation of the existing standard for the United States. There was a high correlation ($r > 0.5$) between PM_{10} particles and elemental concentrations of aluminum, calcium, chromium, iron and silica, which the authors suggested could have played a role in the observed results. No pollutants were found to have robust associations with mortality in Tennessee, although the regression coefficients were positive for most. This lack of association could have been due to exposure missclassification in Tennessee, since the only monitor was located in Harriman, about 50 miles from the main centre of population in Knoxville. In addition, the population of the Kingston-Harriman area was only 640,000, compared to 2.3 million for the St. Louis census area. Both factors would lead to higher variability in results and wider confidence intervals which included the null value, in the case of Tennessee.

Examination of the regression coefficients for the size-fractionated particulate data for St. Louis showed that both $PM_{2.5}$ and the coarse fraction of PM_{10} above 2.5 µm were positively, but marginally ($0.05 < p < 0.1$) associated with mortality, with RR of 1.19 for a 100 µg/m^3 increase in $PM_{2.5}$ ($p=0.075$) and RR of 1.28 ($p=0.056$) for $PM_{10-2.5}$. When considered simultaneously in the regression, it was not possible to demonstrate a more robust association for one than the other, due to high correlations between them. Although sulfates and particle strong acidity, as H^+, showed some positive regression coefficients in St. Louis, these metrics were less predictive of increased mortality than the other particle metrics. The H^+ measurement error was large in St. Louis because concentrations were low and near the quantitation limit. In Tennessee, no pollutants were found to be associated with mortality. This result is noteworthy with respect to H^+, which was present at much higher concentration than in St. Louis (mean 36 µg/m^3 - Tennessee vs. mean 9 µg/m^3 - St. Louis). Overall, in this study, the association of the different particle metrics with mortality was in the order $PM_{10} > PM_{2.5} = PM_{10-2.5} > SO_4^{2-} > H^+$, with PM_{10} clearly the best metric for predicting increases in mortality, followed by $PM_{2.5}$ and the coarse fraction ($PM_{2.5}$-PM_{10}) equally (Dockery et al., 1992).

The data from St. Louis and (Kingston-Harriman) Tennessee and from the remaining four cities in the Harvard Six City Study have recently been re-analyzed, to examine the relationship between daily total mortality and daily changes in various measures of fine and coarse particulate matter (Schwartz et al., 1996a). This study covered eight years between 1979 and 1987 (the previous study had covered only one year). Mean PM_{10} values ranged from 18 to 46 µg/m^3 (overall median 25 µg/m^3 and median of six locations 29 µg/m^3) and mean

$PM_{2.5}$ ranged from 11 to 30 μg/m^3 (median 17.5 μg/m^3). Sampling data were available only every second day in most cases (n=1140 to 1520). For H^+, measurements were available for only approximately one year (n=159 to 429). Poisson regression analyses were carried out, adjusting for temperature, dewpoint and time trends. Pollutants were not considered simultaneously in bivariate analysis because of high correlations between them, nor was lag structure examined in detail because of the incomplete data set.

The strongest and most robust association was found to be with the fine fraction ($PM_{2.5}$); an increase of 10 μg/m^3 in the 2 d mean $PM_{2.5}$ was associated with a 1.5% increase in daily deaths (95% CI 1.1% to 1.9%). The individual city estimates for a 10 μg/m^3 increase in $PM_{2.5}$ (2 d mean) ranged from 0.8% to 2.2% for the six cities. While results were statistically significant for three of the six cities, results for Steubenville and Portage were only marginally significant, and Topeka results were not significant. The association with $PM_{10/15}$ was less marked, with an overall increase of 0.8% (95% CI 0.5 to 1.1%) for each 10 μg/m^3 increase in $PM_{10/15}$ (2 d mean). and with the increases in the individual cities from -0.5 to +1.2% for a 10 μg/m^3 increase (RR=0.95 to 1.12 for 100 μg/m^3). The association was negative for Topeka and positive but not significant (marginal) for Portage. The most interesting results were those concerning the analysis for the coarse mass fraction of particles (CP). In contrast to fine particles or to PM_{10}, CP showed no association with mortality in the overall six-city results, with an estimated increase per 10 μg/m^3 of 0.4% (95% CI -0.1 to 1.0%; t=1.48) In addition, it was also shown to be not associated with mortality in five of the six cities in individual analyses, the one exception being Steubenville, where a 2.4% increase (95% CI 0.5 to 4.3%; t=2.43) was estimated. In this city, the $PM_{2.5}$ result was marginal, and the correlation between the two PM measurements was very high (r=0.69), in contrast to low-moderate correlations for the other five cities (r=0.23-0.43). In simultaneous regressions, coarse particles did not make any independent contribution to the observed association of $PM_{2.5}$ with mortality. In this study, sulphate was also related to mortality, but had a weaker association than $PM_{2.5}$ Particle strong acidity, as H^+, was not related to daily mortality. In this study, the gaseous pollutants were not addressed because of relatively high correlations with PM and the probable confusion that could result during the particulate metrics analysis (Schwartz et al., 1996a).

Daily mortality and air pollution in Toronto, Ontario from 1972-1990, was examined in a preliminary report by Ozkaynak et al. (1995). PM_{10} concentrations (mean 40 μg/m^3) were estimated from TSP, CoH, SO_4^{2-}, and some limited PM_{10} monitoring. Ozone, SO_2, NO_2, and CO were also considered. Multiple regressions were conducted, after detrending the mortality and the air pollution data to remove the effect of seasonal, meteorological and day-to-day variations that were unrelated to air pollution (for example, the effect of Mondays in increasing the official recording of deaths). Either TSP or PM_{10} explained about 2.3% of the deaths that occurred. The relative risk per 100 μg/m^3 increase in PM_{10} was 1.07 (95% CI 1.05 to 1.09) in a single-pollutant regression. Ozone was also associated with mortality, with a relative risk per 100 ppb increase of 1.05, which gave an estimate of 1.5% as the contribution to mortality at the ozone mean of 36 ppb. Thurston (1996) gave further details of the results for PM_{10} of multi-pollutant simultaneous regression, in which the relative risk for PM_{10} was reduced only slightly from 1.07 to 1.05 (95% CI 1.03 to 1.07). The results of this study were therefore consistent with other similar studies in pointing to the independent effects of particulate matter on mortality, but could not distinguish the effects of any one particle measurement.

Dockery et al. (1996b) have recently examined a small mortality data set for Philadelphia, for the summers of 1992 and 1993. Of the PM metrics examined, $PM_{2.5}$ was the most strongly and consistently associated with total and cardiovascular mortality. A 2% increase in mortality per 10 μg/m^3 $PM_{2.5}$ was calculated (95% CI 0.4 to 3.5%; $p<0.01$), while the increase associated with the coarser particle fraction PM_{10} was considerably less, at 1.2% (95% CI not given, but p=0.06, i.e., marginal). No association was found for H^+, SO_2, or CO. A positive association (0.9% per 10 μg/m^3) was found for O_3, which was independent of, and additive to $PM_{2.5}$.

Although a very small study, this adds to the number of results showing that the $PM_{2.5}$ fraction is the one most strongly associated with mortality and other adverse health effects.

12.2.3 Acute Mortality Studies with PM_{10} and BS

There is some uncertainty inherent in comparison of PM_{10}, a gravimetric measurement, with Black Smoke or British Smoke Shade (BS), an optical-based measurement, used commonly in Europe. BS is generally considered to represent a carbon-based dark fraction intermediate in size between PM_{10} and $PM_{2.5}$, with a cutoff size of about 7 μm and a median of 4.5 μm aerodynamic diameter (D_a) (UK Dept. of Environment 1996; US EPA 1996). Moreover, other light-coloured particles such as sulphates, nitrates or acid aerosols would not be included, and would therefore be underestimated, although they may be of importance for adverse health effects (Walters et al., 1994; Anderson et al., 1996). Several recent European studies have included both PM_{10} (or sometimes PM_{13}, which is considered very close to PM_{10}) and BS as particle metrics, thus enabling a limited comparison with results for BS.

Air pollution and its relationship to total mortality was examined in Amsterdam, the Netherlands (population 700,000) for the seven year period 1986-1992 (Verhoeff et al., 1996). Pollution levels were considered relatively low at this time, with mean BS of 12 μg/m^3 (90th centile 23 μg/m^3 and maximum 81 μg/m^3) and mean PM_{10} of 38 μg/m^3 (90th centile 67 μg/m^3 and maximum 163 μg/m^3). Meteorological and temporal variables as well as influenza epidemics were considered in the models. When each was considered separately in the model, both the finer particle measure (BS) and PM_{10} (lag 0 or current day measures for both) were associated with increases in mortality, but BS appeared to have a slightly greater magnitude of association than PM_{10} when considered over their respective interquartile ranges (IQR). The increase per 10 μg/m^3 BS (also the interquartile range (IQR) for this pollutant) was 1.9% (95% CI 0.2 to 3.8%) The increase per 10 μg/m^3 PM_{10} was 0.6% (95% CI -0.14 to +1.4%), and represented a somewhat smaller increase of 1.3% (95% CI -0.3 to 3.1%) for the IQR range of 22 μg/m^3. The relative risk associated with BS was reduced slightly when a one day lag was considered, but the risk associated with PM_{10} was considerably reduced, to about 27% of the original magnitude. In univariate analysis, ozone (1 h max) was also associated with increased mortality when lagged 2 d, with a 0.5% increase (95% CI 0.1 to 1.0 %) per 10 μg/m^3 increase, and a 1.9% increase (95% CI 0.04 to 3.9%) for an IQR increase of 39 μg/m^3. SO_2 and CO were also considered separately, but were not found to be important predictors of mortality. (NO_x was not considered).

When BS or PM_{10} were considered in bivariate analyses separately with with each of the other three gaseous pollutants in turn, BS had a more robust association than PM_{10} with mortality, and the RR for BS remained the same or was increased over the univariate RR, but was reduced for ozone, SO_2 and CO. The association of PM_{10} appeared to be unstable when the three gaseous pollutants were included, and its RR was reduced somewhat with ozone and SO_2, but increased with CO in bivariate analyses. Overall, this study gives support for particulate matter as the best predictor among the air pollutants SO_2, CO or ozone, of an adverse effect of air pollution on mortality. For ozone, the RR remained almost the same as in the univariate analysis at 0.5% (for 10 μg/m^3) when regressed with PM_{10}, but was reduced somewhat to 0.3% when regressed with BS. These results gave some suggestion of an additional association of ozone with mortality that was independent of the association with particulate matter or other air pollutants.

The results of this study were consistent with the findings of other studies that finer particles are more strongly associated with adverse health outcomes than larger particles, based on the more robust association of BS with mortality than PM_{10} when each was considered in bivariate analysis with the individual gaseous pollutants, and on the somewhat greater magnitude of association of BS (1.9%) than PM_{10} (1.3%) with mortality when each was considered with respect to its IQR increase.

As part of the European APHEA collaborative air pollution study, Dab et al. (1996) examined acute mortality from respiratory disorders. In Paris, France (population 6.1 million), over a period of six years, 1987 to 1992, with respect to associations with BS and PM_{13} (measured by radiometry and stated (Zmirou et al. 1996) to be essentially identical to PM_{10}). (See also hospitalization analysis from this study in section 12.3.3) The mean (24 h) BS was 32 μg/m^3 (5th to 99th centile 11-23 μg/m^3) and the mean (24 h) PM_{13} was 51 μg/m^3 (5th to 99th centile 19-137). Data were analyzed using Poisson regression models, adjusted for meteorological variables (mean daily temperature and relative humidity), season, influenza epidemics, weekly and daily patterns and holidays. An increase of 10 μg/m^3 in PM_{13} (lagged by 1 d) was associated with a 1.7% increase in daily deaths (95% CI 0.4 to 3.1%) while a 10 μg/m^3 increase in BS (also lagged 1 d) was associated with a 0.7% increase (95% CI -0.25 to 1.8%). These corresponded to an increase of 8.6% in mortality for an increase in PM_{13} equal to the mean of 51 μg/m^3, and an increase of 2.3% for an increase in

BS equal to the mean of 32 μg/m^3. No conversion factors were provided to relate BS to PM_{10}, PM_{13} or TSP, but a factor of 0.63 between means of BS (~PM_{5-7}) and PM_{10} would give approximately 0.45% increase for a 6.3 μg/m^3 increase in BS compared to a 1.68% increase for a 10 μg/m^3 increase in PM_{10}. Thus, in this location, PM_{13} appeared to be a much better particulate matter predictor than the smaller measure of BS for increased respiratory mortality. The lower association found for BS than for the larger size fraction PM_{13} may reflect the lack of suitability of BS in this location for capturing the non-coloured fraction of fine particles secondarily produced from vehicle combustion, as suggested by Anderson et al. (1996).

SO_2 (1 h maximum, lagged 1 d) was also associated with increased respiratory mortality when analyzed separately in the basic model, with a 0.85% increase in mortality (95% CI 0.15 to 1.6%) for a 10 μg/m^3 increase, or about half that seen for the same increase in PM_{13}. In separate analyses, neither 1 h nor 8 h ozone (lagged 0 d) was significant as a mortality predictor, and 24 h mean NO_2 and SO_2 (both lagged 1 d) were also not significant. The robustness of the BS, PM_{13} or SO_2 mortality associations were not tested by considering SO_2 in bivariate analyses with either of the particulate measures. Respiratory mortality was not associated with any particular season, and individual pollutants, including ozone, were not regressed in seasonal analyses, although ozone was three to four times higher in summer than in winter, and has been shown in some studies (Burnett et al., 1994) to demonstrate associations only in the warm season.

Air pollution and mortality in Athens, Greece (population >3 million) has been investigated in several reports (Katsouyanni et al., 1990; Touloumi et al., 1994) and is included as part of the European APHEA collaborative study (Touloumi et al., 1996). Although BS was the only PM measurement, a site-specific analysis and conversion factor for the BS —> PM_{10} metric was made available (Katsouyanni, 1996; to L. Grant, US EPA, reported in Thurston 1996). Over the five year 1987-1991 period, mean BS was 84 μg/m^3 (range 9-333 μg/m^3) and 42% of days exceeded the WHO air quality guideline. This mean corresponds to a PM_{10} mean of 78 μg/m^3 and a maximum of 306 μg/m^3. SO_2 and CO measurements were also available, and were also relatively high, with means of 51 μg/m^3 for 24 h SO_2 and 6.6 μg/m^3 for 8 h CO. Meteorological (double quadratic function of temperature with a changing point at 22°C, plus relative humidity), seasonal and other daily and long-term variables were adjusted for in the modelling.

A 10 μg/m^3 increase in BS (lagged 1 d) was associated with a 0.5% increase in mortality (95% CI 0.3 to 0.8%). This was calculated to be equivalent to 0.54% (95% CI 0.3 to 0.87%) for an equivalent increase in PM_{10} (based on the formula $PM_{10} = 8.70 + 0.832$ BS). However the strongest association was with SO_2 (lagged by 1 d) and a 10 μg/m^3 increase was associated with a 1.2% increase in total mortality (95% CI 0.7 to 1.6%). CO (current day) was also associated with increased mortality, and a 10 μg/m^3 increase gave a 1.0% increase in mortality (95% CI 0.5 to 1.5%). The estimated increases in mortality were greatest for SO_2 (10%), followed by BS (8%) and CO (5%) when considered according to their respective ranges of increase from the 5th to the 95th percentile in Athens (87.2, 150, and 7600 μg/m^3 respectively).

When BS was included in a bivariate regression with SO_2 the regression coefficient for BS was reduced and that for SO_2 was increased, indicating that SO_2 was the better predictor of the two. The RR for BS was reduced from 1.05 to 1.03 when SO_2 was <100 μg/m^3 and to 1.02 (and was no longer significant) when SO_2 was above 100 μg/m^3. High concentrations of BS (>100 μg/m^3) appeared to act synergistically with SO_2 , with the RR associated with SO_2 rising from 1.12 to 1.15 (12 to 15%). CO was not co-regressed with either of the other two measured pollutants in the APHEA analysis for 1987-91, but was co-regressed in the previous analysis for 1984-88. It was found to be no longer significant (Touloumi et al., 1994), although high correlation (r =0.79 for BS/CO) precludes any conclusion in this regard. No data were available on ozone or NO_2. Air concentrations of all three pollutants BS, CO and SO_2 were highest in late fall and winter, and a significant interaction with season was found for SO_2, but not for BS or CO. The strongest effect of SO_2 was in the winter.

12.2.4 Acute Mortality Studies with BS Only, as the Particle Metric

All-cause, respiratory and cardiovascular mortality and their possible associations with air pollution were investigated in Greater London England (population 8 million), as part of the 14-centre European APHEA collaborative study (Anderson et al., 1996). Particles (BS), ozone, NO_2 and SO_2 (but not CO) were investigated in autoregressive log-linear Poisson regression models, adjusted for time trends (year, season, day of week, bank holidays), seasonal variations, influenza epidemics, and weather (relative humidity and temperature, which was found to have a U-shaped relationship to mortality and was adjusted by using three separate linear terms, for <5°C, 5-20°C, and >20°C). The 24 h average BS was 14.6 $\mu g/m^3$ (10th-90th centiles 8-37 $\mu g/m^3$ and interquartile range 10-18 $\mu g/m^3$).

An increase of 10 $\mu g/m^3$ BS (lag 1 d) was associated with a 1.1% (95% CI 0.55 to 1.72%) increase in all-cause mortality, equivalent to 0.9% (95% CI 0.4 to 1.2%) for an interquartile range increase of 16 $\mu g/m^3$ (reported as 1.7% (95% CI 0.82 to 2.6%) for a 31 $\mu g/m^3$ increase, which was the 10th to 90th centile range). All-year, cool season and warm season BS were all significantly correlated ($p<0.01$) with total mortality when data were subdivided into the cool and the warm seasons. The increases for cardiovascular deaths (CVD) and respiratory mortality were smaller and non-significant, an anomalous finding, not wholly consistent with other studies, and not explainable because of small numbers of daily deaths for these causes, since deaths/day were 78.6 (CVD) and 22.5 (respiratory deaths).

However, the strongest associations with mortality occurred with ozone, when considered as both the 1-h maximum and the 8-h average. A 2.6% increase in all-cause mortality (95% CI 1.3 to 3.8%) was associated with a 10th-90th centile (31 ppb) increase in 1-h ozone, and the increase rose to 3.5% in the warm season (34 ppb increase, the 10th-90th centile range). The all-year, all-cause increase was equivalent to a 0.84% (95% CI 0.4 to 1.3%) increase per 10 ppb ozone, and to 1.3% (95% CI 0.7 to 2,0%) for an interquartile range increase of 16 ppb. The 10th-90th centile increase (24 ppb) in 8 h average ozone gave about the same magnitude of association, 2.4% (95% CI 1.1 to 3.8%), as 1 h ozone with all-cause mortality, and a slightly greater magnitude, 1.0% (95% CI 0.5 to 1.6%) for a 10 ppb increase. Respiratory mortality associated with ozone (1 h max. and 8 h average) were higher than all-cause mortality in the all-year, cool and warm seasons, and respiratory mortality rose to 6.4% for all-year ozone (1 h max) and 6.0% for 8 h ozone. Increases in cardiovascular (CV) mortality were associated in the warm season with increases in both 1 h maximum values and 8 h averages, but all-year CV mortality was asssociated only with 1 h max. ozone. SO_2, which had previously been associated with mortality in the early London Fog episodes (Thurston 1996), was associated with all-cause mortality, but not with CVD or respiratory mortality, in the warm season only (RR 1.52%, 95% CI 0.08 to 2.98% for a 10th to 90th centile increase of 25 ppb). NO_2 (1 h max.) was associated with increases in all-cause, CVD and respiratory mortality in the warm season only, while 24 h NO_2 was associated only with CV mortality, also in the warm season.

In two-pollutant models, both BS and 8 h ozone were found to be independently associated with mortality. The associations with all-cause, respiratory and CV mortality were the same or slightly increased for ozone (8 h) in the all-year and warm-season analyses when BS was included. For BS, all-cause mortality was almost doubled for all year and cool season analyses, and unchanged for warm-season effects when ozone was included in bivariate analyses. The effects of SO_2 and NO_2 were attenuated by inclusion of either BS or ozone in the model, while the latter two both remained significant (Anderson et al., 1996). The effects of CO on mortality and its possible effects on BS and ozone results were not investigated (a re-analysis of the London results including CO has been completed but was not available in time for this document).

The results of this study support an association of daily variations in air pollution with increased mortality. Both ozone and particles, as BS, were shown to have independent associations with all-cause mortality, and summertime ozone with respiratory and CV mortality as well. The magnitude of the associations with ozone were greater than those associated with BS for an equivalent 10th to 90th centile range increase. The associations of either BS or ozone with mortality could not be explained by two other gaseous air pollutants, SO_2 and NO_2, suggesting that particulate matter is the best representative of the three air pollutants arising from primary sources, (i.e., direct combustion) that were examined here.

The acute relationship between air pollution during the six year period from 1985 to 1991, and all-cause, respiratory and cardiovascular mortality in Barcelona Spain was assessed as part of the European APHEA collaborative air pollution study (Sunyer et al., 1996), and also as a supplement to previously conducted morbidity studies (Sunyer et al., 1991, 1993). Results were analyzed using APHEA methodology, with linear regression and autoregressive Poisson models with corrections for temporal variations (year of study, day of week, holidays), seasonal variables, and meteorological factors (linear and quadratic terms for temperature and humidity, terms for high humidity and very hot days). The median number of total deaths was 48/day in winter and 43/day in summer (total number of days 2557). The median 24-h BS was 49.7 $\mu g/m^3$ in winter and 35 $\mu g/m^3$ in summer, and represented one of the higher values in western Europe. SO_2, NO_2 and ozone were also investigated. Most values were calculated for a lag of 1 d.

A 0.7% increase in total mortality was associated with a 10 $\mu g/m^3$ increase in all-year and winter BS (95% CI 0.3 to1.1%). As predicted, the increase was greater for cardiovascular (CVD) and respiratory mortality, at 0. 93% and 0.97% respectively, but was of marginal significance in the case of respiratory mortality, possibly due to increased variance from low numbers of cases (median 5/d compared to 21/d for CV mortality).

The greatest magnitude of association with mortality in all categories was with SO_2 (24 h average 46 and 36 $\mu g/m^3$ in winter and summer respectively). Increases of 1.27% (total mortality, all year) and 1.45% (CVD, all year) were observed per 10 $\mu g/m^3$, although respiratory mortality increases were not significatly associated except in summer (the increase was 2.4%, but note that SO_2 is lower in summer than winter). However, SO_2 was the only pollutant associated with respiratory mortality in this study, (the association with BS being of marginal significance), which agrees with earlier findings for Barcelona on hospitalizations (Sunyer et al., 1991, 1993). The results for SO_2 were also more robust than those for BS in sensitivity analyses incorporating various trends and potential seasonal or weather confounders. The ozone findings were of interest in this study, and contrast to the earlier morbidity studies (ibid.). A 0.48% increase in total mortality and a 0.58% increase in CV mortality were associated with a 10 $\mu g/m^3$ increase in (all year) ozone, while respiratory mortality was not significant, probably for the reason above. The magnitude of the relationship was slightly greater for summertime ozone (median 86.5 $\mu g/m^3$ compared to 55.2 $\mu g/m^3$ in winter). For NO_2, increases were significant for total and CV mortality in all-year and summer analyses, but the increases were not as marked as for SO_2, BS, or ozone.

No pollutants were considered together in bivariate analyses; BS and SO_2 were highly correlated (r=0.63), and thus their effects cannot be separated in regression analyses. The correlations between BS, ozone and NO_2 were weak-moderate (r= -0.33 to 0.29), and the effects of ozone could have been independent. The authors pointed out the possibility of CO exerting a confounding effect, particularly on CV mortality, with which it has been unequivocally associated. The authors also pointed out the weaknesses of using BS as the only surrogate to measure the particle effect, and suggested this as part of the reason for the weaker association of BS with mortality than SO_2. This study also examined whether the mortality was predomintly in the elderly, over age 70 years, or in a younger age group. There was little evidence that overall, total mortality was increased in the elderly by any of the pollutants (reduced for BS and ozone; slightly increased , <0.1%, by NO_2 and SO_2, except 0.2% for SO_2 in winter, all for 10 $\mu g/m^3$ increase) (Sunyer et al., 1996).

The relationship of air pollution, measured as BS and SO_2 was also examined for the three year period 1991-93 in Valencia, a second APHEA centre in Spain (population 750,000) with a warm Mediterranean climate similar to Athens, but with low concentrations of air pollutants, primarily due to vehicle combustion (Ballester et al., 1996). Analyses were undertaken with core models selected by least squares analysis incorporating corrections for season, temperature and humidity, long term trends, day-of the week and holiday effects, and influenza epidemics. Poisson regression was used for each pollutant in separate analyses with two different lag times and warm and cold seasonal adjustments. The mean BS was 67.7 $\mu g/m^3$, and was slightly higher (77.4 $\mu g/m^3$) in winter and lower (58.0 $\mu g/m^3$) in summer. Mean SO_2 was 39.9 $\mu g/m^3$, and did not vary much from winter to summer.

A positive association was found between BS (lagged 3-4 d) and total, CVD or elderly ($\geq$ 70 y) mortality, the RR for a 10 $\mu g/m^3$ increase was 0.9% (95% CI 0.3 to 1.5%) for total mortality, virtually the same for elderly mortality, and 1.2% (95% CI 0.3 to 2.2%) for CV mortality. There was no association with respiratory mortality, likely due to the small number (1.59 deaths/d) of respiratory deaths compared to larger numbers per day (17.5, 11.7, 6.9/d) for total mortality, elderly mortality, or CV mortality. The association with SO_2 was less clear; the relative risk per 10 $\mu g/m^3$ SO_2 over the entire year for total mortality was 0.7% (95% CI -0.3 to 1.5%). SO_2 during the warm months was however, associated with increased CV mortality and with total mortality in the elderly. Introduction of an interaction term between BS and SO_2 in the form of a dummy variable for "low" versus "high" exposure to BS below and above 100 µg/m^3, did not have any effect on the results for SO_2. Pollutants were considered in univariate analyses in order to avoid collinearity, but no correlation coefficients were given (Ballester et al., 1996). These results indicate a positive relationship between air pollution and mortality. This relationship appeared to be more stable for BS than for SO_2 in this locality, and was more similar to results from North American cities than from other European cities. Since no measurements were available for any air pollutants other than BS and SO_2, no further conclusions on which air pollutant is the best indicator of the air pollution effect can be made from these results.

12.2.5 Summary of Results on Acute Mortality

Nineteen studies which investigated the relationship of daily variations in mortality with particulate air pollution using modern time-series methods were reviewed in detail. These have been summarized in Table 12.2. In all of the 15 North and South American locations, studies that used inhalable particulate matter less than 10 µm in diameter (PM_{10}) were selected. Seven recent studies also investigated other, finer fractions, as $PM_{2.5}$ and as sulphate particles. Six European locations measured BS, a fine particle measure with a mean diameter less than 4.5 µm, and in four locations, PM_{10} or PM_{13} (very similar to PM_{10}) was used, sometimes in addition to BS. Most of these studies also examined one or more gaseous pollutants in addition to particulate matter.

Virtually all these studies demonstrated consistent associations between air pollution and acute mortality. These associations could not be explained by the influence of weather (temperature and humidity were most commonly found to have independent associations with mortality), season, yearly trends, day-to-day variations and variations due to holidays, epidemics, or other non-pollutant factors, since all the analyses looked for some or all of these potential biases and accounted for them in various ways in the analysis.

Overall, particulate matter, as PM_{10}, $PM_{2.5}$, BS, and/or sulphate, was clearly associated with all-cause mortality in most of the analyses. These associations were found in North America in 13 locations across the continent, in two locations in large South American cities, and in seven locations across Europe, and showed remarkable consistency with each other despite the widely varying climatic conditions and pollutant mixtures encountered in these cities. In addition, despite a wide range of mean PM_{10} concentrations for these communities, from 18 µg/m^3 (Portage WS) to 115 µg/m^3 (Santiago Chile), the RR range was narrow, indicating highly consistent results under differing PM_{10} exposure conditions. While five of the 23 PM_{10} locations (Salt Lake County, UT, Kingston-Harriman, TN, Topeka, KN, Portage, WS, and Lyon, France) did not show a positive association of PM_{10} with acute all-cause mortality, and marginal associations were found in a further six analyses on all PM metrics (Philadelphia and one of the Utah valley results with $PM_{10,}$ three of the same cities for $PM_{2.5}$ as for PM_{10}, and in Paris, France with BS), the small size of the population studied and/or the low number of "events" (deaths) included (Salt Lake, Kingston-Harriman, Topeka, Portage, Utah Valley) as well as inexact exposure measurements (the earlier study in Kingston-Harriman, and probably others as well) could have been responsible for some of these inconclusive results.

For PM_{10}, the relative risks for mortality from all causes except accidents (calculated for a 100 µg/m^3 increase) varied from 0.95 to 1.17, with a mean of 1.082 ± 0.056 and a median (n=23) of 1.08. The RR were therefore on the low end of the range previously reported (Dockery and Pope 1994; Schwartz 1994e; Pope et al., 1995c) of 1.07 to 1.16 for each 100 µg/m^3 PM_{10} increase. For the fine fractions $PM_{2.5}$, and BS, the unweighted mean relative risks were also elevated with concentration means (1.15 (n=9) and 1.10 (n=6) respectively). Only one study provided a combined quantitative estimate for sulphate for six US cities, and the mean RR (100 µg/m^3 increase) was 1.22. These results mean that an increase in daily deaths of about 0.8% can be predicted for each 10 µg/m^3 daily increase in $PM_{10,}$, and a 1.0% and 1.5% increase for similar size increases in BS and $PM_{2.5}$. While this increase is of very low magnitude, it signifies substantial numbers of avoidable deaths due to the very large size of the population that is impacted by air pollution.

Table 12.2 Summary of Relative Risks for Particulate Matter: Total Mortality

LOCATION	PM METRIC (MEAN; RANGE)	RR (100 ug/m³ increase) (95% CONFIDENCE LIMITS)	RR + OTHER POLLUTANT IN MODEL (95% CI) (95% Confidence Limits with other pollutant in model)	REFERENCE
St. Louis, US	PM_{10} (28; max 97)	1.16 (1.01 – 1.30)	1.12 (0.96 – 1.15) (+O_3) (US EPA 1996)	Dockery et al., 1992
Kingston-Harriman US	PM_{10} (30; max 67)	1.17 (0.88 – 1.50)	1.17 (0.88 – 1.52) (+O_3) (")	Dockery et al., 1992
Utah Valley US	PM_{10} (47; 1-365)	1.16 (1.10 – 1.22)	1.14 (1.04 – 1.24) (+O_3) (Pope & Kalkstein 1996)	Pope et al., 1992
Philadelphia US	PM_{10} (not given)	1.12 (0.99-; upper CI not given) (p=0.06)	—	Dockery et al., 1996b
Birmingham US	PM_{10} (48; 21-80, max 163)	1.11 (1.02 – 1.20)	—	Schwartz 1993
Toronto Canada	PM_{10} (40; not given in text)	1.07 (1.05 – 1.09)	1.05 (1.03 – 1.07) (+ "other pollutants") (Thurston 1996)	Ozkaynak et al., 1995
Los Angeles US	PM_{10} (58; 15-177)	1.05 (1.00 – 1.11)	1.05 (1.00 -1.11) (+O_3) 1.04 (0.98 – 1.09) (+ CO)	Kinney et al., 1995
Chicago (Cook Co.) US	PM_{10} (38-median; 28-51 IQR)	1.055 (1.01 – 1.10) (>65 y)	—	Styer et al., 1996
Chicago (Cook Co) US	PM_{10} (41 +19)	1.05 (1.03 – 1.08)	1.04 (1.01 – 1.07) (+O_3)	Ito and Thurston 1996
Utah Valley US	PM_{10} (47: 1-365)	1.04 (0.98 – 1.10)	—	Lyon et al., 1995
Salt Lake Co US	PM_{10} (48: (9-194)	1.008 (~0.8 – 1.08) (> 65 y)	—	Styer et al., 1995
6-city study, (combined)	PM_{10} (25; 8-68)	1.08 (1.05 – 1.11)	—	Schwartz et al., 1996a
St. Louis (US)	30.6 ± 14.5	1.06 (1.01 – 1.10)		
K-H, Tenn.	24.5 ± 12.8	1.09 (1.01 – 1.18)		
Boston	45.6 ± 32.3	1.12 (1.07 – 1.17)		
Steubenville	26.7 ± 16.1	1.09 (1.01 – 1.16)		
Topeka	17.8 ± 11.7	0.95 (0.80 – 1.09)		
Portage		1.08 (0.96 – 1.17)		
Sao Paulo Brazil	PM_{10} (82 + 39)	1.13 (1.07 – 1.19) (>65 y)	1.13 (1.03 – 1.23) (+O_3, SO_2, NO_2, CO)	Saldiva et al., 1995
Santiago Chile	PM_{10} (115; 32-367)	1.07 (0.99 – 1.11) (OLS, summer) 1.08 (1.06 – 1.12) (Poisson, all year)	1.06 (1.00 – 1.09) (+O_3, OLS, summer) 1.04 (1.01 – 1.08) (+ SO_2, Poisson, all year) 1.07 (1.03 – 1.11) (+ NO_2, Poisson, all year)	Ostro et al., 1996
Paris France	PM_{13} (51: 19-137)	1.17 (1.04 – 1.31)	—	Dab et al., 1996
Lyon France	PM_{10} (38: 5-98)	1.02 (0.94 – 1.10) total deaths-accidents (1.04; 1.02 – 1.09 respiratory deaths)	RR for PM n.g. with SO_2	Zmirou et al., 1996
Athens Greece	PM_{10} (78; 8-300)	(1.063 (1.033 – 1.09) – estimate from BS)	[1.03 (1.01 – 1.06) (SO_2<100) – estimate from BS] [1.02 (0.97 – 1.07) (SO_2>100) – estimate from BS]	Touloumi et al., 1996

Abbreviations: RR – relative risk; C.L. -confidence limits; IQR – interquartile range; n.g. – not given; OLS – Ordinary Least Squares regression;

Table 12.2 Summary of Relative Risks for Particulate Matter: Total Mortality *(continued)*

LOCATION	PM METRIC (MEAN; RANGE)	RR (100 ug/m^3 increase) (95% CONFIDENCE LIMITS)	RR + OTHER POLLUTANT IN MODEL (95% CI) (95% Confidence Limits with other pollutant in model)	REFERENCE
Amsterdam NL	PM_{10} (38; max 163)	1.06 (0.99 – 1.14)	1.03 (0.94 – 1.13) (+O_3) 1.02 (0.93 – 1.13) (+ SO_2) 1.10 (1.01 – 1.20) (+ CO)	Verhoeff et al., 1996
Unweighted rel. risk: PM alone (n=23) mean 1.082 ± 0.056 (95% CI range 0.80 – 1.50) median 1.08 ; bivariate analysis. PM + other gases (n=16): median 1.05; mean 1.069 ± 0.047				
Amsterdam NL	BS (12; max 81)	1.19 (1.02 – 1.38)	1.18 (1.01 – 1.37) (+O_3) 1.27 (1.07 – 1.49) (+ SO_2) 1.20 (1.01 – 1.44) (+ CO)	Verhoeff et al., 1996
London England	BS (15; 8-22, 10th-90th percentile)	1.11 (1.05 – 1.17)	1.19 (1.11 – 1.) (+O_3)	Anderson et al., 1996
Barcelona Spain	BS (50,35; winter, summer)	1.07 (1.03 – 1.11)	—	Sunyer et al., 1996
Valencia Spain	BS (68 + 29)	1.09 (1.03 – 1.15)	—	Ballester et al., 1996
Paris France	BS (32:11-123; 5th-95th percentile)	1.07 (0.98 – 1.18)	—	Dab et al., 1996
Athens Greece	BS (84; 9-333)	1.05 (1.03 – 1.08)	1.03 (1.01 – 1.06) (SO_2<100) 1.02 (0.97 – 1.08) (SO_2>100)	Touloumi et al., 1996
Unweighted mean relative risk 1.096 ± 0.050 (95% CI range 0.98 – 1.38) median 1.08 (95% CI 1.03 – 1.16)				
St. Louis, US	$PM_{2.5}$ 17.7 (max 75)	1.19 (p=0.075) (95% CI not given	—	Dockery et al., 1992
Kingston-Harriman US	$PM_{2.5}$ 21 (max 58)	1.17 (p=0.28) (95% CI not given)	—	Dockery et al., 1992
Philadelphia, US	$PM_{2.5}$ (not given)	1.20 (1.04 – 1.35)	RR for PM + O_3 not stated (both independent)	Dockery et al., 1996b
6-city study, (combined)	$PM_{2.5}$ 14.7 (4.3-431)(IQR 14.0)	1.15 (1.11 – 1.19)	"same" (+ CP or $PM_{10-2.5}$)	Schwartz et al., 1996a
St. Louis (US)	18.7± 10.5	1.11 (1.04 – 1.17)	CP – RR=1.24 (1.05-1.43) correlation=0.69	
K-H. Tenn.	20.8 ± 9.6	1.14 (1.02 – 1.26)		
Boston	15.7 ± 9.2	1.22 (1.15 – 1.29)		
Steubenville	29.6 ± 21.9	1.10 (0.99 – 1.21)		
Topeka	12.2 ± 7.4	1.08 (0.80 – 1.36)		
Portage	11.2 ± 7.8	1.12 (0.97 – 1.28)		
Unweightled mean relative risk (6 individual cities + 3 cities) 1.148 ± 0.049 median 1.14 (95% CI range 0.8 0- 1.36)				
St. Louis Mo. US	SO_4^{2-} 8.0 (max 38)	weak pos., n.s.	—	Dockery et al., 1992
Kingston Tenn. US	SO_4^{2-} 8.7 (max 27)	n.s.	—	Dockery et al., 1992
6-city study US (combined)	SO_4^{2-} 5.8 (1.5-22.3)	1.22 (1.13 – 1)	SO_4^{2-} RR strong. (t=4.66) but less than $PM_{2.5}$ (t=7.41)	Schwartz et al., 1996a
Too few data points for comparison				

Abbreviations: RR – relative risk; CI -confidence limits; IQR – interquartile range; n.g. – not given; OLS – Ordinary Least Squares regression:

These increases in relative risk due to air pollution were observed at particulate matter concentrations substantially below the current international and Canadian 24-hour objectives/standards. Canada currently has objectives only for TSP, as a 24-h acceptable level of 120 µg/m^3 , approximately equivalent (depending on location) to PM_{10} of 60-80 µg/m^3 and to $PM_{2.5}$ of about 30-50 µg/m^3. In the majority of the studies examined the mean/median (24 h) concentrations of PM_{10} were below 50 µg/m^3 (median of 22 locations 43 µg/m^3), and $PM_{2.5}$ means were below 20 µg/m^3 (median of 8 locations 18 µg/m^3). The lowest mean concentrations at which PM_{10} mortality associations were positive were in Boston MA at 24.5 µg/m^3, and in the overall results for six cities (including Boston) at 25 µg/m^3 PM_{10} and 14.7 µg/m^3 $PM_{2.5}$ (Schwartz et al., 1996a).

There was little evidence in these data that the dose-response curve included a threshold, and the response was observed to increase monotonically (in the concentration range below 80-100 µg/m^3) with increasing concentration (all PM_{10} data) in both nonparametric analyses (Birmingham AB – Schwartz 1993; Santiago – Ostro et al., 1996; Utah Valley – Pope and Kalkstein 1996) and quartile or quintile analyses (Utah Valley – Pope et al., 1992; Birmingham, AB – Schwartz 1993; Sao Paulo, Brazil – Saldiva et al., 1995) The quintile results for St. Louis, MO and Kingston, TN (Dockery et al., 1992) were also roughly monotonic when taken together, but not separately, due to relatively poor exposure information and (in Tennessee) to low population. A curvilinear response, with high slope at low concentrations, leveling off at higher concentrations, was observed above 100 µg/m^3 in Santiago, Chile, and possibly also in the Birmingham data, above about 50 µg/m^3. Although the quintile analysis of Utah Valley by Lyon et al. (1996) indicated a definite threshold at the mean for quintile 4 of 47 µg/m^3, discrepancies were evident in the quintile means, and caution was indicated in interpretation of these data. The lack of a threshold down to low concentrations suggests that it will be difficult to select a level at which no adverse effects would be expected to occur as a result of exposure to particulate matter.

There was evidence from the six-city acute mortality study that the fine fraction of particulate matter ($PM_{2.5}$) was consistently associated with mortality, and that the association was of greater magnitude than that with PM_{10} or sulphate for increases equivalent to the IQR for each (2.1% for $PM_{2.5}$ versus 1.8% for PM_{10} and 1.5% for SO_4^{2-}). Moreover, the coarse fraction of PM_{10} above $PM_{2.5}$ in diameter was not associated with mortality in the overall analysis (0.4%; 95% CI -.01 to 1.0% for IQR), or in the five individual cities in which the correlation coefficents between fine and coarse fractions were below 0.46 (the association was positive in the one city with a high correlation coefficient of 0.69). This association of the fine fraction was supported by results from a small study in Philadelphia and by one of the two European studies which measured both BS (relatively fine) and PM_{10} or PM_{13} (Dockery et al., 1996b; Dab et al. 1996). Sulphate, which forms a part of the fine fraction, has been suggested as the best index for fine particle effects (Lippmann and Thurston 1996), but the results from the six-city study do not support this hypothesis when both measures are available, since the magnitude of effect for $PM_{2.5}$ was substantially greater than the magnitude of effect for sulphate for an equivalent concentration range. Moreover, the US EPA analyzed the non-sulphate portion of $PM_{2.5}$ separately from sulphate and found that the relative risk was as high or higher for the non-sulphate fraction as for the sulphate fraction (US EPA 1996).

The elderly and those with pre-existing respiratory and cardiovascular diseases have been suggested as being at increased risk from exposure to particulate matter (Schwartz 1993, 1994d; Lipfert 1994). A slight increase (13%) in relative risk from 1.5 to 1.7% for a 10 µg/m^3 increase in $PM_{2.5}$ was observed for those aged ≥ 65 years in the six-city daily mortality study (Schwartz et al., 1996). The RR for the elderly aged ≥ 70 years was virtually unchanged (an 11% decrease) in Barcelona and Valencia Spain (Sunyer et al., 1996; Ballester et al., 1996), using BS as the particle metric. In Santiago, Chile a 43% increase in the RR (per 100 µg/m^3 increase) over the all-cause RR was observed for the elderly ≥ 65 years, while in the Lyon et al. (1995) analysis for the Utah Valley and in the Styer et al. (1995) results for Chicago, IL, the risk appeared to be doubled for those over age 65. The increases in RR for the various age groups in the Lyon study appeared to be unstable, probably because of the low number of respondents (n=4036), but this was not the explanation for the Chicago study, which presented detailed results for deaths in the fall season only. Overall, the results suggest only a surprisingly modest increase in relative risk for the elderly compared to the whole population. This does not support the suggestion that it is only the elderly who are being affected and dying from air pollution, and that their lives are being shortened by air pollution episodes only by a few days or weeks before they would have died anyway.

A number of investigators looked at the specificity of the air pollution effect on the causes of mortality. Deaths from respiratory disease were moderately to strongly elevated compared to all-cause deaths in the six-city daily study, and in Chicago, IL, Birmingham, AB, Santiago, Chile, Barcelona, Spain and Lyon, France (Schwartz 1993; Schwartz et al., 1996; Ostro et al., 1996; Sunyer et al., 1996; Zmirou et al., 1996). Only in three studies, in London England (Anderson et al. 1996), Valencia Spain (Ballester et al., 1996) and in the Lyon et al. (1995) analysis for the Utah Valley, was a decrease noted in RR for respiratory deaths compared to total mortality. In the latter two locations, the few deaths per day could have contributed to the lower non-significant RR, but this could not have been the explanation for London. In the six-city study, both chronic obstructive pulmonary disease (COPD) and pneumonia were found to exhibit strongly increased relative risks over the total mortality RR. For COPD, the risk was almost doubled, and for pneumonia, the risk was 2.7 times the RR for total mortality of 1.5% for a 10 µg/m^3 increase in PM$_{2.5}$

The association between air pollution and cardiovascular disease was also clear. Modest increases over the total mortality RR were noted in the relative risk for death due to cardiovascular diseases in the six-city study (RR increased from 1.5% to 1.7% for a 10 µg/m^3 increase in PM$_{2.5}$), Barcelona, Spain (RR increased from 0.7 to 0.9%) and in Valencia, Spain (RR increased from 0.9 to 1.2% for a 10 µg/m^3 increase in BS). Substantial increases were observed in Birmingam, AB (55%) and the Utah Valley (300%) (Schwartz 1993; Lyon et al., 1995). The RR declined from the total mortality RR in only two locations, Chicago (RR for a 10 µg/m^3 increase in PM$_{10}$ decreased from 0.5 to 0.3%) and in London, England (RR decreased from 1.7 to 0.6%) (Ito and Thurston 1996; Anderson et al., 1996). Overall, the association with cardiovascular disease was well marked but was only modestly increased by comparison to total mortality. Moreover, the association with cardiovascular disease was often of lesser magnitude than the association with respiratory disease, particularly in all the large cities, (six-city study, Chicago, IL, Santiago, Chile) with the exception of London, England.

Particulate matter is not the only air pollutant that could be associated with increases in mortality. Because of the strong intercorrelations often demonstrated between the primarily produced air pollutants PM, SO$_2$, NO$_2$, and CO, as well as correlations between SO$_2$, NO$_2$, and secondarily produced fine PM as sulphates and nitrates, it has been difficult or impossible for any one study to attribute the air pollution effect to any single one of these agents to the exclusion of others. A bivariate or multipollutant regression model treats each variable as independent, an assumption that is invalid if these variables are highly correlated with each other as they often are. The best method available thus far has been examination of single pollutant models in regression analyses which tried to account for all non-pollutant-related factors and any potential confounders such as temperature before examining the relationship of the pollutant to mortality. If positive relationships with several pollutants were found, bivariate analyses could then be carried out, bearing in mind that the validity of the results would depend (inversely) on the degree of correlation of the two pollutants. Some investigators have also looked for interrelationships between pollutants by examining the effects of one pollutant at high and low levels of the other pollutant, for example the effect of SO$_2$ above and below 100 µg/m^3 on the association of BS with mortality in Athens (Touloumi et al., 1996). Many investigators examined at least one other air pollutant in addition to PM, in order to determine if any other air pollutant was a better predictor of increased mortality than was PM, or was independently associated with an increased relative risk. The results of bivariate analyses with SO$_2$, NO$_2$, CO and ozone on the estimates of relative risk associated with PM$_{10}$ and BS are shown in Table 12.2. In the only study which quantitatively examined fine particles as PM$_{2.5}$ and sulphate, no bivariate regressions were undertaken because none of the other pollutants were associated in single analyses, and there was potential for correlation problems as well (Schwartz et al., 1996b).

Overall, although the particulate matter coefficients were slightly reduced, they retained their association with acute mortality in bivariate analyses (and in one preliminary multivariate analysis for Toronto (Ozkaynak et al., 1995)). The mean and median RR for PM$_{10}$ (per 100 µg/m^3 increase) was reduced from 1.08 (n=23) to 1.05 (n=16).The mean and median RR for BS of 1.10 and 1.08 respectively (n=6) rose to a mean of 1.15 and median of 1.19 in three analyses with SO$_2$, two with ozone and one with CO, but this result is biased upward by consideration of three of six results from a single location and study in which relative risks were estimated to be high for BS with or without the inclusion of other air pollutants in the analysis (Verhoeff et al., 1996).

The possible relationship of SO_2 with mortality was considered for 14 locations, since it had in the past been associated with wintertime peaks in PM and episodes of mortality and morbidity (the London Fogs and other episodes of the 1950s). In general, while SO_2 appeared to be the best predictor of air pollution effects on mortality in a few European locations, in most locations its effects were attenuated in regressions which included a particulate matter metric, and in several locations, it did not appear to be associated with mortality even in single pollutant analyses. However, it should be noted that a number of studies investigating TSP (not reviewed here) have also reported positive associations with SO_2. Sulphur dioxide was the air pollutant most strongly associated with mortality in Lyon, France, Athens, Greece and Barcelona, Spain, remaining significantly associated in Lyon and Athens after inclusion of PM_{13} in Lyon (Zmirou et al., 1996) and BS in Athens (Touloumi et al., 1996). A high correlation coefficient with BS (0.63) precluded pairwise comparision in Barcelona (Sunyer et al., 1996), and calls for caution in interpretation of the results of the other two studies also, with respect to the role of SO_2 (the PM-SO_2 correlation coefficient varied with season between -0.08 and +0.76 in Lyon, and was reported in an earlier paper (Touloumi et al., 1994) as "substantial" in Athens). In single-pollutant regression analyses, SO_2 was found to have positive associations with mortality in Santiago, Sao Paulo, Paris, London and Valencia (Ostro et al., 1996; Saldiva et al., 1995; Dab et al., 1996; Anderson et al. 1996; Ballester et al., 1996), but pairwise analyses with PM_{10}, BS or both attenuated the effects of SO_2 while having little effect on the PM coefficients. SO_2 was not associated with mortality in univariate analyses in Chicago IL (Ito and Thurston 1996), St. Louis, MO and Kingston-Harriman, TN (Dockery et al., 1992), Philadelphia, PA (Dockery et al., 1996b), Toronto (Ozkaynak et al., 1995) and Amsterdam (Verhoeff et al., 1996). Thus, particulate matter is considered to be more robust predictor of the air pollution-mortality effect compared to SO_2, in the great majority of locations.

Nitrogen oxides (usually NO_2) were examined in nine locations. Overall, the evidence for an association of NO_2 with mortality is considered weak. In four locations (Lyon and Paris, France, St. Louis, MO and E. Tennessee US) no association between mortality and NO_2 was found in single pollutant analyses. In Sao Paulo, Brazil and Santiago, Chile, the association of NO_2 with mortality in the single-pollutant model was reduced to non-significance in bipollutant models, while the coefficient for PM_{10} remained high. Correlation coefficents between NO_2 and PM_{10} were high in both locations, 0.55-0.68 in Sao Paulo and 0.73 in Santiago. NO_2 was also not associated with mortality in Toronto in a multi-pollutant analysis (no single-pollutant results were presented) (Ozkaynak et al., 1995). In London, England, NO_2 (1 h maximum) was associated with increased risks due to cardiovascular disease, in summer only. The relative risks associated with (1 h) NO_2 were smaller and less consistent than for BS or ozone, and (1 h) NO_2 was not included in bivariate analyses in this study (Anderson et al., 1996). No association was found for 24 h NO_2 (Anderson et al., 1996). In Barcelona, NO_2 (1 h max.) was associated with total and CV mortality as well as with mortality in the elderly $\geq$ 70 years old. No pairwise regression with other pollutants were undertaken, although the correlation coefficients for BS-O_3-NO_2 were relatively low at 0.29-0.30. The authors suggested a confounding effect with (unmeasured) CO, in view of the CVD increases rather than respiratory increases as might have been expected from what is known of the pulmonary toxicity of NO_2 (Sunyer et al., 1996). Particulate matter, as BS or PM_{10}, was the air pollutant most strongly and consistently associated with mortality in all these studies except in London, England, where the strongest associations were with ozone (Anderson et al., 1996), and in Barcelona, Spain, where the air pollutant which best predicted increases in mortality was SO_2 (Sunyer et al., 1996).

Carbon monoxide (CO), which is a primary vehicular combustion product like PM and NO_2, was considered in two European and five North and South American locations. CO was associated with mortality in Los Angeles, CA and in Athens, Greece. In Los Angeles, the regression coefficients for both CO and PM_{10} were reduced only slightly in bivariate regresssions, which could indicate that each was a good, independent predictor of the air pollution effect on mortality, but no correlations between them were given (Kinney et al., 1995). In Athens, the increase associated with CO was 10% (95% CI 5 to 15%) for a 10 $\mu g/m^3$ increase, compared to 12% for SO_2 and 5% for BS. Based on equivalent 5th-95th percentile range increases, the increase associated with CO was 7.6% (95% CI 3.8 to 11.4%), about the same as with BS and lower than SO_2 at 10.5%. The BS-CO correlation coefficient was 0.79, too high for bivariate regressions to be undertaken (Touloumi et al., 1996). CO was not associated with all-cause mortality in Chicago, Philadelphia or Amsterdam when regressed singly (Ito and Thurston 1996; Dockery et al., 1996b; Verhoeff et al., 1996), or in Sao Paulo or Toronto in multiple-pollutant regressions (Saldiva et al., 1996; Ozkaynak et al., 1995). Although particulate matter was the best index of air pollution in these five cities, further work is warranted on the possible association of CO with mortality, in view of the high correlations between them which could obscure any relationship, and of the biological plausibility of a link between CO and cardiovascular disease.

The possibility of an association between ozone and acute mortality was examined in 13 locations. Ozone toxicity is known to occur after short periods (one to six hours) of exposure, and ozone concentrations are also most frequently highly cyclic in a diurnal pattern. Therefore the results for ozone are often more sensitive than particles or other air pollutant gases to the duration of the measurement, with 1 h maximum and 5 to 8 h daytime values for ozone more likely to be more strongly associated with adverse health than 24 h values, which tend to smooth out the effect of diurnal peaks. The manner of its secondary formation via photochemical oxidation of primary combustion products means that it is also highly seasonal, with highest concentrations observed in summer. Therefore an analysis on a yearly basis is less likely to demonstrate associations than an analysis on a seasonal basis. However this type of formation also means that it is usually not highly correlated with particulate matter, making it easier to detect independent associations. These factors were taken into account in weighing the evidence for an effect of ozone on mortality that was independent of other air pollutants.

The relationship of ozone, as one of several air pollutants, to acute mortality was examined in 13 locations. Ozone (1 h daily maximum) was associated with mortality in single-pollutant regression models in 8 of these cities: London, England (also the 8 h daily average), Barcelona, Spain, Amsterdam, NL, Santiago, Chile, Chicago, IL, Philadelphia, PA, Toronto, ON and Los Angeles, CA (Anderson et al., 1996; Sunyer et al., 1996; Verhoeff et al., 1996; Ostro et al., 1996; Ito and Thurston 1996; Dockery et al., 1996b; Ozkaynak et al., 1995; Kinney et al., 1995). The relative risks on a yearly basis ranged from 0.2 to 2.6% for a 10 ppb or a 10 $\mu g/m^3$ increase. Separate analysis for the cool and warm seasons in London, England and Santiago, Chile demonstrated that risks were higher for summertime than when the entire year was included (Anderson et al., 1996; Ostro et al., 1996). In all these cities except Barcelona, two-pollutant analyses were carried out with the inclusion of both particulate matter and ozone in the model. In Los Angeles, there was a reduction to a RR of 1.0 for ozone and no change in the RR for PM_{10} based on only 364 days of simultaneous measurement (Kinney et al., 1995). In the remaining seven cities, the relative risks for both ozone and PM remained the same or were slightly, but not greatly, reduced in magnitude and the confidence limits widened. Thus both ozone and particulate matter (PM_{10} in most locations, BS in London and Amsterdam) appeared to act as independent factors in the association of air pollution with daily mortality. This conclusion is strengthened by the fact that the correlations between ozone and PM were very low to low-moderate, ranging from -0.29 to +0.37 in five of these cities. No association of total daily mortality with ozone was found in single-pollutant models in five additional cities: Sao Paulo, Brazil, Lyon and Paris, France, St. Louis, MO and eastern Tennessee, US (Dockery et al., 1992; Saldiva et al., 1995; Zmirou et al., 1996; Dab et al., 1996). In the two US locations, 24 h measurements and poor placement of monitors with respect to the place of residence of most of the potential responders could have been partly responsible for the negative results for ozone. No obvious reason for the lack of association of ozone was evident in the other three cities, for which both 1 h and 24 h monitoring results were available. In 10 of the 13 locations that investigated the relationship of mortality to ozone in additon to other air pollutants, particulate matter appeared to have the strongest association with mortality of the several air pollutants that were examined. The exceptions were London, England, where the RR for an increase in ozone equal to the 10th to 90th percentile range was found to be 2.4% (95% CI 1.1 to 3.8%), compared to 1.7% (95% CI 0.8 to 2.6%) for BS (Anderson et al., 1996), Barcelona, Spain and Lyon, France where SO_2 was the best predictor of increased mortality compared to BS and ozone (Barcelona) or PM_{13} and ozone (Lyon) (Sunyer et al., 1996; Zmirou et al., 1996). The weight of evidence is here considered to be relatively strong that ozone has an association with daily mortality that is independent of the PM association.

In all of the analyses that examined one (or more) air pollutants together in the same model with particulate matter, the association of particulate matter with daily mortality was remarkably robust, despite the problems of disentangling the effects of PM from other air pollutants. This was the case for all four of the normally considered gaseous pollutants, SO_2, NO_2, CO and ozone. Moreover, in most locations the magnitude of the PM association was greater than any other air pollutant considered, the exceptions being ozone in London and SO_2 in Lyon and Barcelona. The magnitude, robustness and consistency of this association across so many locations with differing air pollutant mixtures indicates that PM is the best indicator of the air pollution effect on mortality, and is considered to give some support to PM of some kind, possibly acting together with other air pollutant(s) as a causal agent. An attempt to address the question of which metric of particulate matter might be the best indicator for the "PM effect" was made in one study, which provided strong evidence that it was the fine fraction ($PM_{2.5}$) that was most closely associated with mortality.

12.3 ACUTE EFFECTS – HOSPITALIZATIONS AND EMERGENCY DEPARTMENT VISITS

A number of studies, mostly longitudinal time-series analyses of acute or short-term effects, have examined hospital admissions for respiratory disease as an indicator of health, and a few have examined cardiovascular outcomes as well. The data presented show a wide range of geographical locations and particulate sources. The studies which included only PM_{10} as the particle metric have been considered separately from studies which examined the relationship of hospitalizations to various measures of fine particles, often in addition to PM_{10}.

12.3.1 Hospital Studies and PM_{10}

Pope (1991) examined the association between monthly respiratory admissions and PM_{10} air pollution for three populated areas in the state of Utah: the Utah Valley, (population 188,000); the Cache Valley (population 72,500) and the Salt Lake Valley (population 780,000) over the four year period between April 1985 and March 1989 (Pope 1991). Annual mean PM_{10} concentrations were 53, 55, and <40 μg/m^3 for Utah Valley, Salt Lake and Cache Valley respectively, and were highest in winter, at 95, 71 and <60 μg/m^3. The main source of particulate pollution in the Utah Valley was a steel mill, which contributed approximately 50 to 70% of the PM_{10} pollution in this area. This steel mill only slightly affected PM_{10} levels in the Salt Lake Valley, about 60 km to the north. Sources of PM_{10} pollution in the Salt Lake Valley were varied. Particulate levels in the Cache Valley were generally low, so admissions data from this location were used as a reference level or control. Overall levels of SO_2 and NO_2 were said to be low and in compliance with EPA standards, but, as demonstrated by other studies with PM, this is no guarantee that associations with adverse health would not be found. Although no quantitative data were presented, SO_2 was reported to be near the limit of detection, and ozone was low in winter when PM_{10} concentrations peaked. Smoking rates were also low. Hospital admissions were recorded for pneumonia, bronchitis and asthma. Total respiratory admissions in these categories were 4144 for Utah Valley, 7373 for Salt Lake, and 1468 for Cache Valley. Admissions were regressed using ordinary least squares (OLS) estimates with autoregressive models using maximum likelihood estimation (statistical references given). A trend variable for each month of study, monthly mean low temperature, PM_{10}, and lagged PM_{10} and temperature were regressed.

Average hospital admissions (all ages) for pneumonia, asthma and bronchitis were higher in the Utah Valley when the steel mill was open than when it was closed for 13 months. The rates of admissions were 1.17, 1.40 and 1.14 for pneumonia, bronchitis and asthma respectively, and were higher than those in the Cache Valley (RR= 0.95, 0.92 and 1.24, the latter of marginal significance). The preschool age group was identified as a potential susceptible subgroup. When the steel mill was open, ratios for bronchitis and asthma were twice as high for children aged 0-5 years (RR= 2.09 and 2.01 for bronchitis and asthma respectively). These differences were not observed in either the Salt Lake or the Cache Valley. During periods when the steel mill was in operation, the ratio of hospitalization rates for bronchitis or asthma in the Utah Valley was twice as high in the Utah Valley as in the Cache Valley (Pope 1991). While this study is not strictly comparable to daily time-series studies since it examined monthly changes, it provides the results of an interesting "natural experiment" in which hospitalization rates were demonstrated to be reduced when the source of much of the PM_{10} pollution was removed, i.e., a cause-effect relationship was demonstrated on a population basis. The cross sectional comparison within the design acted to provide a control population and confirmed this rise in rates. An additional point of interest was the much stronger effect on preschool children than on the population as a whole with respect particularly to bronchitis and asthma. The results of this study are highly consistent with the hypothesis that particulate matter pollution, as here measured by PM_{10}, plays an important role in the incidence and severity of respiratory disease.

A daily time-series was conducted in the Seattle area comparing daily PM_{10} measurements with hospital emergency department visits for asthma, with gastroenteritis as a control diagnosis, in eight hospitals over a 13 month period from September 1, 1989 to September 30, 1990 (Schwartz et al.,1993). Measurements were also collected for SO_2 and O_3 (no data were presented for O_3, and the length of the monitoring period was also not stated). The maximum and mean PM_{10} concentrations were 103 $\mu g/m^3$ and 29.6 ± 18 $\mu g/m^3$, respectively. PM_{10} levels were measured from a residential site in a wood-burning area where wood smoke (fine particulate matter) was thought to be the main source of the particulate pollution. Ozone was measured from a site 20 miles east of the city, which would likely have had higher levels than the city itself, due to NO_x scavenging in the area with more vehicular traffic. There were 2955 visits for asthma during the study period (mean 7.1/d).

Results showed a highly significant association between PM_{10} concentrations and asthma admissions, at low environmental levels. A relative risk (RR) of 1.12, (95% CI 1.04-1.20), was calculated for an increase equal to the mean of 30 $\mu g/m^3$ PM_{10}. This estimate was robust to differences in model specifications for temperature adjustment, relative humidity, or for cold dry days. Division of the results by quartile of PM_{10} demonstrated a dose response, with RR increasing from the second (mean 20 $\mu g/m^3$) to the fourth quartile (mean, 30 and 55 $\mu g/m^3$) by comparison to the first (mean 13 $\mu g/m^3$) (RR and confidence intervals not presented except as a graph). No associations were found for gastroenteritis, or for the other air pollutants examined, SO_2 and ozone (RR=0.99 and 0.97 respectively). However, the association with light scattering (km^{-1}), which is a measure of fine particles less than 2.5 µm, was of the same magnitude as PM_{10}, with a RR of 1.11 (95% CI 1.04-1.18 for a change of 7 km^{-1} equivalent to the change in PM_{10}) (Schwartz et al., 1993). Despite the small size of this study (one year, relatively few visits), it suggests that particulate air pollution from the small size fraction produced by wood smoke may play a role in exacerbation of asthma. The investigation of other air pollutants which could also have been associated with respiratory illnesses was weak, especially for ozone, which has been shown in other studies to have such an association independently from PM_{10}.

In Birmingham, Alabama, daily counts of hospital admissions for the respiratory illnesses pneumonia and chronic obstructive pulmonary disease (COPD) were recorded in the elderly (≥ 65 y) for the five years 1986 to 1989 inclusive (Schwartz 1994a). The admission means were 5.9/d for pneumonia and 2.2/d for COPD. Twenty-four-hour PM_{10} pollution data were collected at three monitoring sites for 1369 (94%) of the possible days throughout the study period. The mean PM_{10} level for the study period was 45 $\mu g/m^3$, (10th to 90th percentile range: 19 $\mu g/m^3$ and 77 $\mu g/m^3$). Ozone (1 h peak levels and 24 h average) was also considered. Data were analyzed in Poisson regression models controlling carefully for yearly trends, season, and weather. For pneumonia, the relative risk calculated for a same-day PM_{10} increase of 100 $\mu g/m^3$ was 1.19 (95% CI 1.07-1.32). The relative risk for COPD was 1.27 (95% CI 1.08-1.50). It was also calculated that inhalable particles were responsible for 8% of the pneumonia admissions and 12% of the COPD admissions at the mean concentration of 45 $\mu g/m^3$. Results were not sensitive to several different methods of controlling for seasonality or weather, or to extremes of temperature. In a quartile analysis of the same data, a monotonic response was observed, with no evidence for a threshold from 13 to 55 $\mu g/m^3$ (means for quartile 1 to quartile 4). A similar yet weaker significant relationship was found with COPD admissions. It was also calculated that inhalable particles were responsible for 8% of the pneumonia admissions and 12% of the COPD admissions at the mean concentration of 45 $\mu g/m^3$. Ozone was weakly associated with pneumonia admissions two days later; the relative risk for a 50 ppb increase in 24 h ozone was 1.14 (95% CI 0.94-1.38) and for 1 h peak ozone was 1.04 (95% CI 0.97-1.12). Peak ozone was also weakly associated with COPD admissions; the relative risk was 1.07 (95% CI 0.96-1.20). No attempt was made to assess the interactions or the independence of the associations with PM_{10} and ozone in bivariate regressions, although the correlation coefficients between them were relatively low (r=0.29). The correlation between ozone and temperature was also negligible (r=0.04) in this city.

Using data from hospitals in Detroit, Michigan (total population 4 382 000 in 1990, population base 517 000 for those age 65 and older), daily admissions for three categories of respiratory diseases (pneumonia, chronic obstructive pulmonary disease (COPD) including asthma, and asthma alone: means 15.7/d, 5.8/d and 0.75/d respectively) were recorded for the four years 1986 to 1989, for persons aged 65 and older, and compared with daily pollution data available through various monitoring stations in Detroit (Schwartz 1994b). Pollution variables included ozone as well as PM_{10}. PM_{10} levels ranged from a 10th percentile of 22 $\mu g/m^3$ to a 90th percentile of 90 $\mu g/m^3$, with a mean of 48 $\mu g/m^3$. After control for season and weather (temperature and dew point), associations were found in bivariate regressions between both PM_{10} and ozone and hospital admissions for pneumonia. The relative risk for pneumonia with concurrent-day PM_{10} levels was 1.012 (95% CI 1.004-1.019 for a 10 $\mu g/m^3$ increase in PM_{10}). The relative risk of pneumonia for a 5 ppb increase in 24 h ozone was 1.026 (95% CI 1.015 to 1.04). When analyzed by quartile of PM_{10} and ozone, a linear dose response was evident for PM_{10}, the RR increasing from 1.0 at 22 $\mu g/m^3$, (quartile 1) to 1.075 at a mean of 88 $\mu g/m^3$ (quartile 4). The dose response was not so evident for ozone, and the RR was not increased until quartile 3 (mean ozone about 25 ppb) Both PM_{10} and ozone were significantly associated with COPD admissions (other than asthma) as well. The increased risk for PM_{10} and COPD (RR = 1.020, 95% CI 1.004-1.032 for a 10 $\mu g/m^3$ increase) was higher than for pneumonia. The RR for ozone in the same pairwise model was 1.03 (95% CI 1.007-1.045 for a 5 ppb increase) The inclusion of both PM_{10} and ozone in the same model did not appreciably lower the coefficients for either pollutant with either endpoint (data not shown), and the correlation coefficient between them was 0.35, or moderately low. These results suggested that both were independently associated with respiratory disease. Sensitivity analyses indicated that extremes of high or low temperature or the methods of controlling for seasonality were not responsible for the associations observed with PM_{10}. The associations seen with 24 h ozone were also seen with 1 h maximum ozone but the effect was weaker. Neither PM_{10} nor ozone were associated with increases in asthma (Schwartz, 1994b), possibly because of the low number of daily admissions recorded for this disease, and also because this disease is less prevalent than COPD or infectious diseases in older persons. This study provides evidence that both PM_{10} and ozone are independent risk factors for respiratory disease, and that the associations, at least for PM_{10}, are not due to bias introduced by the treatment of seasonal or weather effects.

The association between daily counts of hospital admissions for cardiovascular disease in the elderly was also examined, again using Detroit data during the four years 1986 to 1989 (Schwartz and Morris 1995). Admissions categories examined included ischemic heart disease (44/d), dysrhythmias (13/d) and congestive heart failure (27/d). The same pollution data set was used as described in the above study, with the addition of data on SO_2 and CO. After careful control for temperature, dewpoint, seasonal and day-to-day variations, PM_{10} (mean of current and previous day) was found to be a significant predictor of admissions for ischemic heart disease (IHD) in single pollutant regressions (RR = 1.056, 95% CI = 1.016-1.10), as well as for admissions for congestive heart failure (CHF) in a bivariate regression with CO (RR = 1.075, 95% CI 1.013-1.14) (both relative risks calculated for a 100 $\mu g/m^3$ increase based on relative risks, for an interquartile increase of 32 $\mu g/m^3$, of 1.018 (95% CI 1.005-1.032) for IHD and 1.024 (95% CI 1.004-1.044) for CHF.) A curvilinear dose-response was shown for quartile analysis of the results for IHD and PM_{10}, with steeply rising slope at low concentrations (approximately 22 to 36 $\mu g/m^3$) and lower slope at higher concentrations (approximately 55 to 85 $\mu g/m^3$).

Although ozone was not associated with ischemic heart disease (IHD) admissions in single pollutant analysis, associations were observed for SO_2 and CO, but neither SO_2 nor CO remained associated with IHD in bivariate analyses with PM_{10}. CO was strongly associated in single pollutant regression with admissions for congestive heart failure (CHF). In bivariate regressions, the regression coefficients and relative risks remained constant for CO (RR=1.022; 95% CI 1.010-1.034 for IQR increase of 1.28 ppm) and were reduced somewhat from 1.032 to 1.024 for an IQR increase in PM_{10}, indicating an independent association for both pollutants with CHF (CO and PM_{10} correlations were moderately low, at 0.30). The results for PM_{10} for IHD and for both PM_{10} and CO in bivariate analyses were subjected to sensitivity analyses; the results could not be explained on the basis of the extreme temperatures, pollutant levels above the US standards, other temperature lags, or various regression types (robust Poisson regression, Generalized Additive Model with nonparametric smooth functions of time, temperature and humidity).

Data on influenza and other non-air-pollutant-related epidemics were not available and could not be controlled for. Little association was observed between PM_{10} levels and admissions for dysrhythmias. (Schwartz and Morris, 1995).

This study was one of the first to show associations between PM_{10} air pollution and cardiovascular disease, in addition to respiratory disease outcomes that had previously been shown for this city (Schwartz 1994b) and elsewhere. It also provides evidence that ozone is not associated with cardiovascular disease, and supports the hypothesis that it is associated specifically with respiratory disease. A major finding was the association between CO and admissions for congestive heart failure; these associations, for both CO and PM_{10}, were found to be independent of each other, and the results were robust to alternate methods of estimation and weather control.

Hospital admissions for respiratory illnesses in the elderly (aged 65 years and older) and PM_{10} levels were examined in the metropolitan area of Minneapolis-St. Paul, Minnesota (population 2.46 million in 1990) for the four years 1986 to 1989 (Schwartz 1994c). Mean admissions were 6/day for pneumonia and 2/d for chronic obstructive pulmonary disease (COPD). Mean PM_{10} concentrations were 36 $\mu g/m^3$ and the interquartile range was 23 to 44 $\mu g/m^3$. Ozone (24 h average) was also examined. After control for time trends, season, and weather in Poisson regression analyses, the results for models including both pollutants showed that current-day PM_{10} levels were significantly associated with hospital admissions for pneumonia (RR=1.17, 95% CI 1.03-1.33 for a 100 $\mu g/m^3$ increase). Ozone had a similar relationship (RR=1.15; 95% CI 0.97-1.36 for a 50 ppb increase), which changed from marginal to significant when a different temperature control was used. When both pollutants were considered simultaneously, both were considered to have made independent contributions to explaining pneumonia hospitalizations, but the ozone result were not as consistent after sensitivity analysis for the effects of extreme temperature, high pollutant levels or model specification. The correlation between PM_{10} and ozone was low, 0.18, but the correlation between ozone and temperature was high, 0.51, making it difficult to separate out the effects of temperature from ozone. For COPD hospital admissions, both concurrent and previous-day PM_{10} levels were again significantly associated with hospital admissions (RR=1.22, 95% CI 0.99-1.52 and RR=1.37, 95% CI 1.12-1.68, respectively, for 100 $\mu g/m^3$ increases), the effect being greater with a one-day lag. A weighted average of the current and previous day's PM_{10} levels captured the sum of both effects on COPD (RR=1.57, 95% CI 1.20-2.06). Ozone (24 h and 1 h maximum) was not associated with COPD in this study. Seasonal patterns, weather conditions, misclassification of disease and high air pollution episodes were all reported to have been controlled for. The association found with hospital admissions for pneumonia and ozone, but not COPD and ozone (Schwartz 1994c) is consistent with results from previous studies in Birmingham and Detroit by the same author (Schwartz, 1994a,b).

Daily counts of hospital admissions for respiratory disease for people aged 65 years old and older during three years from 1988 to 1990, were compared to average daily concentrations of PM_{10}, SO_2, and O_3 in two cities, New Haven, Connecticut and Tacoma, Washington (Schwartz 1995). Mean respiratory admissions were 8.1/d in New Haven and 4.2/d in Tacoma. Mean, interquartile range, and 90th percentile concentrations for PM_{10} were 41, 25 and 67 $\mu g/m^3$, respectively for New Haven, and 37, 27 and 67 $\mu g/m^3$, respectively for Tacoma. After inclusion of a 19 d moving average filter to remove cyclic variations, and further adjustments for temperature including extremely hot days, humidity, and day of the week effects, results from Poisson regression analyses showed that all three pollutants were positively associated with hospital admissions for respiratory disease in both cities, the most consistent association being with PM_{10}. Both cities were also found to be consistent regarding the lag of two days for the association of PM_{10} with respiratory hospital admissions. Calculated relative risk estimates (and 95% confidence limits) for each increase of 50 $\mu g/m^3$ PM_{10} in single pollutant regressions were 1.06 (1.00-1.13) for New Haven and 1.10 (1.03-1.17) for Tacoma. In New Haven, inclusion of either of the other pollutants with PM_{10} did not reduce the estimated magnitude of either the PM_{10} effect estimate, or the estimates for ozone and SO_2, indicating the strong possibility of independent effects for all three pollutants in this city (no correlation analysis results were given). In Tacoma, the magnitude of the PM_{10} effect was the same or slightly larger in two-pollutant analyses with SO_2 and ozone, similar to New Haven, but in Tacoma, the relative risk for a 25 ppb increase in ozone remained the same in the bivariate analysis (RR=1.20; 95% CL 1.06-1.37) while the relative risk for PM_{10} included 1.0 (RR=1.12; 95% CI 0.97-1.29). In Tacoma, SO_2 was no longer associated in the bivariate analysis that included either PM_{10} or ozone (Schwartz 1995). While no correlations between pollutants were provided, the consistency of the results overall provide some support for an independent association for both PM_{10} and ozone in Tacoma well as in New Haven. On the other hand the SO_2 association was attenuated by ozone in both cities, and by PM_{10} in Tacoma.

Hospital admissions for respiratory disease and air pollution were examined in Spokane, Washington for patients aged 65 and older for the three years 1988 to 1990 (Schwartz, 1996). Mean respiratory admissions were 3.9/d, and the two largest hospital admission subcategories were pneumonia (1.9/d) and chronic obstructive pulmonary disease (1.0/d). Air pollution data were collected for particulates (PM_{10}) every 24 h and for ozone (O_3) on both a 24 h and an hourly basis. The mean level of (24 h) PM_{10} was 46 $\mu g/m^3$, with an interquartile range of 24 to 57 $\mu g/m^3$, and a 10th to 90th percentile range of 16 to 83 $\mu g/m^3$. Results were analyzed using Poisson regression with the Generalized Additive Model, which allowed the fitting of non-linear functions for weather (temperature and dew point), day of study, season and day of the week, and interactions between these variables, with smoothing functions for each that were chosen based on minimization of Aikaiki's information criterion (statistical references given in paper). This type of model had the advantage of not trying to force a linear shape to the variables, which showed non-linearities in data. An association between same-day PM_{10} and respiratory admissions was found, with a relative risk (for a 50 $\mu g/m^3$ increase) for all respiratory categories of 1.085 (95% CI 1.036-1.136), for pneumonia, 1.053 (95% CI, 0.985-1.126) and 1.171 (95% CI, 1.079-1.272) for chronic obstructive pulmonary disease. Peak ozone (1 h maximum) was also associated with an increase in all respiratory admissions (RR=1.24; 95% CI 1.002-1.544 for a 50 $\mu g/m^3$ or 25 ppb increase), but not with the subcategories.The results for PM_{10} were robust to changes in the model to control for temperature, but the ozone results were more sensitive to the handling of temperature. This was not surprising in view of the negligible correlation (0.034) between PM_{10} and temperature, and the moderate ozone-temperature correlation (0.44). While the correlation between ozone and PM_{10} was relatively low at 0.26, no bivariate analyses could be undertaken with both pollutants in the model because there were only 115 days in which both pollutants were measured togther. Nonetheless, the author rightly considered that this study added support to other studies that suggested the existence of an (independent) association (Schwartz 1996). While data were not presented on SO_2, its stated virtual absence in this location precludes confounding by this pollutant. Moreover, the lack of correlation between PM_{10} and temperature suggests that the positive findings for PM_{10} cannot be explained by temperature.

Gordian et al. (1996) studied the association, for the period May 1, 1992 to March 1, 1994 (22 months), between particulate pollution and the incidence of acute respiratory diseases as measured by daily outpatient visits for specific respiratory diagnoses in Anchorage, Alaska, an area without significant industrial pollution. The population of Anchorage is approximately 240 000 and it is located on the seacoast, in a bowl surrounded by mountains. The main sources of particulates were unpaved roads, road sanding, vehicular traffic, and ashfall from volcanic eruptions. The authors stated that wood-burning was not a major source of PM in Anchorage because wood was too expensive to use as a heating fuel. Limited monitoring for $PM_{2.5}$ carried out intermittantly over several years indicated that the fine particle fraction of PM_{10} ($PM_{2.5}$) formed an unusually low portion of the total PM_{10}, the ratio $PM_{2.5}$/coarse fraction of PM_{10} being 0.26 on average, and as low as 0.14 in some years. Daily mean, (plus range) air pollution measurements for PM_{10} and carbon monoxide (winter season only for CO) were recorded as 45.5, (5 to 565) $\mu g/m^3$ and 2.5, (0.5 to 7) $\mu g/m^3$, respectively. Health data were grouped using ICD (International Classification of Diseases) codes to identify upper respiratory problems such as sore throat, earaches, sinusitis, rhinitis, and other nonspecific upper airway problems. The bronchitis group included lower airway diseases such as bronchitis, tracheitis, and nonspecific cough. The asthma group included all reactive airway disease, bronchospasm, and asthma. Diarrhea was selected as a control diagnosis. The number of people eligible for the study was approximately 14 000 workers and their families employed by the state of Alaska and the city of Anchorage, who were covered by a single health insurance company. Claims for emergency visits were analyzed using both Poisson and multiple linear regression analyses, the latter detrended using a 19 d moving average (Shumway filter), adjusted for long-term cyclic variations, seasonal effects, day of the week effects, and temperature.

Researchers found statistically significant associations between either same day or previous day PM_{10} and outpatient visit for illnesses due to asthma and upper respiratory illness (URI), but no association with bronchitis after adjustment for several factors. The increase was about 3.5% for asthma and 1.2% to 2.7% for URI, both for a 10 $\mu g/m^3$ increase in PM_{10}, in a location where the primary sources of PM_{10} were not combustion or secondary aerosols. However, the association found between PM_{10} and asthma-related doctors' visits was not significant during the period that was affected by volcanic activity from Mt. Spur, 60 miles west of Anchorage (Gordian et al., 1996). This study thus gave mixed signals regarding the importance of the particle fraction between $PM_{2.5}$ and PM_{10} ("coarse particle" (CP) fraction), and indicated that silicaceous or volcanic crustal coarse particulate pollution may have less than expected adverse acute respiratory effects, but that the urban CP fraction may also be associated with adverse repiratory effects , even in a healthy, young, working population. Wintertime CO was also found to be associated with outpatient visits for bronchitis and upper respiratory tract infections, but not with asthma. These associations might have been explained by the high correlation between CO and automobile exhaust emissions, but models including both PM_{10} and CO produced results essentially the same as the single pollutant regression models. However, due to the small number of days on which CO was measured, the authors advocate caution in interpretation of the CO results. Only sporadic measurements were available for SO_2, NO_2, and ozone. However, the authors considered it unlikely that any of these could have been responsible for the associations since SO_2 measurements were less than 10% of the US EPA standard, and NO_2 and ozone measurements were less than 33% of that standard. This argument is open to question considering the results of others showing adverse health outcomes at very low levels of pollutants. Overall, the most intriguing findings from this study have to do with the effects of a particulate mass composed largely of coarse particles, >2.5 µm.

In three communities in eastern Washington State (1990 population 101 623), the number of Emergency Department visits in 1991 for respiratory disorders such as otitis media, bronchitis, upper respiratory tract infections, pharyngitis, sinusitis, croup, pneumonia, asthma, influenza, bronchiolitis, and chronic obstructive pulmonary disease were compared to daily PM_{10} levels (Hefflin et al., 1994). Dust storms are a major source of PM_{10} in Washington State, the particulates being coarse, volcanic in origin, and belonging to the plagioclase (glass) mineral class of aluminum silicates and other oxides. The coarse fraction ($PM_{10-2.5}$) thus makes up a greater proportion of PM_{10} than usual. The total number of emergency visits for all respiratory disorders was 23 349 or 64/d. For a total of 278 daily PM_{10} samples, the daily mean value was 40 $\mu g/m^3$, with a daily range of 3-1,689 $\mu g/m^3$. The results showed a relationship between PM_{10} levels and the number of emergency room visits during 1991 for bronchitis and sinusitis. No association was found between PM_{10} and asthma despite the expected prevalence of asthma in such a community. A 10 $\mu g/m^3$ increase in PM_{10} was associated with a 0.35% increase in visits for bronchitis ($p=0.03$). In October 1991, two dust storms occurred, during which PM_{10} levels went above 1000 $\mu g/m^3$ on several days. Although the odds ratio for bronchitis was raised to 1.2 in the dust storms, this was less than expected on the basis of the high PM_{10} levels experienced, thus indicating that crustal dust is less toxic than finer particles, which are usually produced as a result of combustion processes.

12.3.2 Hospital Studies and TSP

The relationship between pollution levels and hospital emergency room visits were studied in the industrial town of Steubenville, Ohio, during the spring months of March and April, and fall months of October and November during 1974-1977 (Samet et al., 1981). Records of emergency room visits covering 10 categories (asthma, emphysema and/or chronic bronchitis, pneumonia, lower respiratory infection, upper respiratory infection, other respiratory infection, other respiratory disease, cardiac disease. cardiac and respiratory disease, trauma, and miscellaneous) were collected and compared to air pollution data. Monitored pollutants included total suspended particles (TSP), sulphur dioxide (SO_2), nitrogen dioxide (NO_2), carbon monoxide (CO), and ozone (O_3). The 24 hour average was utilized as the index of pollution exposure. The TSP mean was measured at 156 $\mu g/m^3$ with a range of 14-696 $\mu g/m^3$. Analysis of the resulting data showed no consistent correlation of lagged and unlagged pollutant levels with either total emergency room visits or with visits for diagnoses other than trauma (Samet et al., 1981). Since this was an early study, the negative results might have been attributed to the less rigorous statistical treatment given to the data, by comparison to later time-series analyses, but a later re-analysis, by the original author, of these data using contemporary statistical methods yielded the same results as the original (Samet 1997, personal communication.) The negative results could also have been due to use of the TSP metric, which has a coarse particle size cutoff above the inhalable range, and thus does not provide as close a match to the real exposure of individuals in the population as a smaller size fraction.

The effects of low levels of pollution and weather conditions on the number of patients admitted to hospital for exacerbation of chronic bronchitis or emphysema were studied in Helsinki, Finland during a three-year period (1987-1989) (Ponka and Virtanen 1994). In addition to total suspended particulates (TSP), other air pollutants monitored included SO_2, NO_2, and O_3. The main source of air pollutants in the Helsinki area is coal-fired and oil-fired power plants, road traffic, and industrialization. Pollution data collected were daily mean concentrations and the ranges, the results for TSP being 76 µg/m^3 and 6-414 µg/m^3, respectively. Final analysis showed no significant effect of either temperature or relative humidity, or of TSP or O_3 on the daily number of hospital admissions. However, it was also explained that approximately one-third of the TSP air pollution data were missing, and therefore had to be estimated (Ponka and Virtanen, 1994). The previously described study excluded any hospital admissions for asthma. A separate report was published using the same air pollution data on the association between asthma and ambient air in pollution in Helsinki (Ponka and Virtanen 1996). No association was observed between asthma and TSP levels. However, associations were observed between some of the other air pollutants and asthma. Due to some unexplained results, the method of analysis was deemed unsatisfactory as was the collection of data for TSP, (Ponka and Virtanen, 1996).

The above studies were all conducted after an initial examination of the association between asthma and air pollution by the same author in the Helsinki region (Ponka 1991). Hospital admissions were examined over a three-year period between 1987 and 1989. During this period, 4,209 admissions to hospital for acute asthma were recorded. Pollutant levels were reported for NO_2, NO, SO_2, CO, O_3, and TSP. Mean TSP levels were 76.3 µg/m^3 with a range of 6.0-414.0 µg/m^3. Admissions were increased 28%, 29%, 15% and 18% associated with periods of high NO, NO_2, CO, and TSP pollution respectively, compared to periods of lower pollution. No clear cut limits could be observed on the basis of health effects, but it was observed that as pollution increased so did health effects. It should also be noted that it was not the incidence of asthma that was considered in this study, but rather, the frequency of acute attacks among persons with pre-existing bronchial asthma that was recorded (Ponka, 1991).

12.3.3 Hospitalization Studies with Various Fine Particle Measures

Thurston et al. (1994) conducted a daily time-series in the city of Toronto, Ontario, (population 2.5 million) utilizing measured TSP, PM_{10}, $PM_{2.5-10}$, $PM_{2.5}$, SO_4^{2-}, H^+, O_3, SO_2 and NO_2 and evaluating the association with daily respiratory effects-related hospital admissions for 22 Metropolitan Toronto-area hospitals. Monitoring was carried out for six weeks in July and August of the years 1986-1988, (n=40-42 days x 3 years). The 24 hour average PM_{10} level ranged from 30 to 39 µg/m^3, wih a maximum of 86 µg/m^3, $PM_{2.5}$ ranged from 15.8 to 22.3 µg/m^3, with maximum values ranging from 50.7 to 66.0 µg/m^3, and SO_4^{2-} means were 74, 38 and 124 nmol/m^3 (7.1, 3.6 and 11.9 µg/m^3) for 1986-88 respectively. When regressed singly, ozone, H^+, fine particles ($PM_{2.5}$), PM_{10} and sulfate were all associated with hospital admissions; for a 10 µg/m^3 increase in SO_4^{2-}, $PM_{2.5}$, and PM_{10} , respiratory hospitalizations increased 8.0% (0-16%), 3.2% (-3.0 to 9.3%) and 4.7% (0.5-8.8%) respectively (95% CI in brackets). Coarser particles between 2.5 and 10 µm in diameter, and TSP were found to have weaker marginal associations with hospital admissions, and association with the very coarse fraction above PM_{10} to be non-existent (associations with SO_2 and NO_2 were also non-significant). When each particle metric was regressed simultaneously with ozone, only H^+ remained significant (p=0.04), and sulfate was also marginally significant (p=0.09). Regression coefficients for all PM fell 20-50% in the bivariate regressions and the ozone coefficient remained about the same (with H^+) or was reduced 20-25%. Because of high intercorrelations (r=0.64-0.73) between ozone and all PM pollutant measures except H^+ (moderately high), the authors considered that it was not possible to establish whether ozone and the particle metrics had independent effects or whether any one pollutant was the best index of the adverse health effects of air pollution. However, the results suggested that particle strong acidity was an important contributor to respiratory admissions, at least in this location. It was also observed that associations generally became stronger as particle size decreased (i.e., TSP < PM_{10} < $PM_{2.5}$) and that chemical composition of the particle mass (acidity and SO_4^{2-}) had an influence on daily summertime respiratory and asthma hospital admissions.

In a well conducted study on summertime respiratory hospital admissions, the daily number of unscheduled respiratory admissions was recorded for 168 acute care hospitals in southern Ontario (population 8.7 million, or over 90% of the entire population of the province) for the six years between 1983 and 1988 (Burnett et al., 1994). Mean respiratory admissions were 107.5/d. The only particle metric available over the entire area was sulphate. The yearly mean was 4.37 $\mu g/m^3$ (range 1-31 $\mu g/m^3$) and the warm period (April-September) mean was 5.13 $\mu g/m^3$. Mean annual ozone (1 h maximum) was 36 ppb and the summertime mean was 50 ppb. Data were analyzed by multiple regression, taking account of day-of-week variations in hospitalization rates, inter-hospital variations, and longer or seasonal variations making use of a 19 d weighted filter to remove all cyclic variations in the data for health outcomes and exposure variables.

The investigators found significant associations during the May-August summer period, between hospital admissions (all ages) for asthma, COPD, and infections, and both ozone and sulphates recorded on the day of admission and up to three days previously, with the highest association on the previous day (lag 1 d) for both pollutants. In summer, ozone was more strongly associated than sulphates when the two were included in the same model . Five percent of daily admissions were associated with a 50 ppb increase in ozone while a further 1% could be ascribed to a 5.3 $\mu g/m^3$ increase in sulphates. However, the high correlation between these two (0.38-0.65 among various stations) indicates that caution is needed when assigning effects to each pollutant. When plotted by decile of concentration (lagged one day), the dose-response appeared to increase monotonically from 0 to 20 $\mu g/m^3$ for sulphate and from 20 to 95 ppb for ozone. Other particulate metrics (TSP, PM_{10}, CoH) could not be investigated because daily data were not available, and/or there was insufficient area coverage of the monitoring network. Two other air pollutants (SO_2, NO_2) were considered, but insufficient monitoring stations were available to give monitoring data representative of the entire 900 km long area. Relatively low correlations between SO_2, NO_2 and the two pollutants sulphate and ozone, that were included in the regression analysis provide some assurance that SO_2 and NO_2 were not likely to bias the association for sulphate (Burnett et al., 1994).

Burnett et al. (1995) further analyzed this database with respect to sulphate and other particle metrics estimated from sulfate and expanded the study to include hospital admissions for cardiovascular cases as well as respiratory admissions. Groups of diagnoses thought to be unrelated to air pollutants (gastrointestinal, genitourinary, nervous system, and blood system) were also included as controls. As before, the daily number of unscheduled admissions was recorded for 168 acute care hospitals for the six years between 1983 and 1988. Data were analyzed by multiple regression, taking account of day-of-week variations in hospitalization rates, inter-hospital variations, and longer or seasonal variations making use of a 19 d weighted filter for admission data. Regressions were performed for winter and summer separately, adjusting for temperature and ozone, which were highly correlated with sulphates (for temperature, r=0.35, 0.15, and 0.43 for the year, cool and warm seasons respectively; for ozone, r=0.47, -0.04, and 0.53 for the year, cool and warm seasons respectively) and were considered as potential confounders. Adjustments for temperature and ozone were done separately for the warm and the cool season. Yearly sulphate levels averaged 4.4 $\mu g/m^3$ with a median of 3.1 $\mu g/m^3$; the 95th and 99th percentiles were 13.0 and 23.0 $\mu g/m^3$ respectively. As with ozone, sulphate levels were higher in the warm season (April-September) than in the cold season. The predicted values of PM_{10} based on a sulphate level of 13 $\mu g/m^3$ were 47.0, 52.1 and 44.5 $\mu g/m^3$ for the Toronto, Windsor and Ottawa regions, respectively, (also central, western and eastern limits of the entire area) with an average of 48.0 $\mu g/m^3$, which would represent very well the more than half of the population living in the central region. Note, however, that a concentration of 32 $\mu g/m^3$ was reported in the paper by Burnett et al. (1995, page 21) which could be due to an intercept effect.

For both respiratory and cardiovascular illnesses, hospitalization rates were found to increase with increasing sulphate concentration. A 13 $\mu g/m^3$ increase in sulfate concentration (the 95th percentile) was associated with a 3.7% increase in respiratory admissions (95% CI 2.3 to 4.7%), and a 2.8% increase in cardiac admissions (95% CI 1.7 to 4.8%) ($p<0.0001$ for both). The associations were almost the same after adjustment of ozone, square ozone, temperature, and square temperature: the increase was 3.5% (95% CI 2.3-4.7%) for respiratory admissions and 3.3% (95% CI 1.7-4.8%) for cardiac admissions. The relationship appeared linear for respiratory effects-related admissions; however, the concentration-response appeared to be curvilinear for cardiovascular illnesses (i.e., the increase in rate tended to diminish for cardiovascular illnesses as the sulphate concentration increased. When the total sample was divided into four groups according to increasing SO_4^{2-} concentration, the adjusted daily average hospitalization rate for the lowest half of the sample, with an average sulphate concentration of 1.55 μg/m^3, was statistically different from the corresponding average hospitalization rate for the next higher group (the 25% around the average, at the 4.13 μg/m^3 sulphate concentration) for both respiratory and cardiovascular illnesses ($p < 0.01$).

Positive associations were observed for all age groups, sexes, and time periods for cardiac diseases including coronary artery disease (2.3%) and heart failure (3.0%) (but not dysrhythmias) and respiratory diseases including asthma (3.2%), COPD (4.8%), and infection (3.3%). For respiratory diseases, excess hospitalizations were calculated to be 2.7% for children <15 y, compared to 3.8% and 3.7% for adults 15-64 y and ≥65 y. Cardiac admissions were increased more for the elderly (≥65 years) than for those under age 65, at 3.5% for the former versus 2.5% for the latter. Increases in both respiratory and cardiac admissions were similar in the winter and the summer period, after adjustment for temperature and ozone. The results were robust to application of 15 different regression models (Burnett et al., 1995).

This study clearly demonstrated that sulphates, despite the low levels experienced in southern Ontario (the sample median was 3.07 μg/m^3), are associated with excess hospitalizations for both respiratory and cardiac diseases. Although the correlation between ozone and sulphate was moderately high (r=0.53), these associations can not be explained by ozone, since the effect of sulphates was observed over the entire year as well as in summer, and persisted in winter when the correlation was low. The method of adjustment for temperature also appeared to be satisfactory. A major strength of this study is its size – about one-third of the population of Canada, over a period of six years. Its weakness is perhaps in the lack of ability to consider the role of other measures of particulate matter (PM_{10}, $PM_{2.5}$) or other air pollutants – SO_2, NO_2, CO – some of which could have had high correlations with sulphates as well as associations with increased adverse health outcomes.

Summertime respiratory and cardiac admissions to Metro Toronto hospitals during June to September 1992-94 (388 days) were examined in a preliminary study by Burnett et al. (1997) with respect to possible associations with a suite of particulate matter metrics as well as the four criteria gaseous pollutants. Fine particle metrics included $PM_{2.5}$ (mean 14 μg/m^3; range 4-40 μg/m^3), SO_4^{2-} (mean 33 nmol/m^3 or 3.2 μg/m^3; range 5-229 nmol/m^3 or 0.5-22 μg/m^3), particle strong acidity as H^+ (mean 1 nmol/m^3; range 0-23 nmol/m^3), and coefficent of haze (CoH: mean 0.8 x 10^3 lin. ft.). Coarse particle metrics included CP or $PM_{2.5-10}$ (mean 10 μg/m^3; range 4-23 μg/m^3) and PM_{10} (mean 25 μg/m^3; range 10-58 μg/m^3). Correlation coefficients between the various particle metrics and NO_2 were moderate to high (0.34 to 0.61) except for H^+ (r=0.25). Correlation coefficients with SO_2 were moderate (0.42 to 0.55), and correlation coefficients between particles and ozone were moderately low (0.23 to 0.34) except for SO_4^{2-} (r=0.53).

After adjustment for long term temporal trends, seasonal variations, short term epidemics, day of the week effects and ambient temperature and dew point, positive associations were observed in Poisson regressions carried out individually for each of the PM metrics. For respiratory admissions, relative risks for the observed particulate pollutant interquartile ranges, adjusted for temperature and dew point, were 1.023 to 1.037, all significant. The magnitude of the association was highest for CoH and fine particles and least for H^+ and coarse particles. When regressed pairwise with each gaseous pollutant, the relative risks for all the particle metrics were slightly to moderately reduced, but all retained significance except PM_{10} with NO_2 (for which the correlation coefficient was high, 0.61). Ozone retained its significance, and its magnitude of association was the same or slightly lower in all the pairwise analyses, while the relative risk for CO was reduced to half or less of its univariate value and was not significant with any of the particle metrics. While the relative risks for SO_2 and NO_2 dropped and were no longer significant for regressions with CoH, the RRs dropped more moderately in pairwise regression with the other particle metrics and retained their significance.

For cardiac admissions, all particle metrics except $PM_{2.5}$ and SO_4^{2-} were significantly associated in the single regressions, again with CoH having the highest magnitude of association (RR for CoH =1.062; t=5.6). In the pairwise regressions, CoH retained its association with each of the gases (CoH-gas correlation coefficients were 0.2, 0.3, 0.5 and 0.6 for CO, O_3, SO_2 and NO_2). Relative risks for SO_4^{2-}, PM_{10} and fine particles were no longer significant with any of the gases in bivariate models. The relative risk for H^+ retained its magnitude with NO_2 (r=0.25), and the coarse particle relative risk was increased with ozone (r=0.20); otherwise, the relative risks for H^+ and coarse particles were all reduced to non-significance with NO_2 or SO_2 (H^+ r=0.25-0.45; CP r=0.44-0.57). Ozone was again the only gas that consistently retained significance when regressed with the various PM matrics (Burnett et al., 1996). The negative results for cardiac disease observed for sulphate and fine particles in this much smaller summer-only study contradict the previous findings of the much larger multi-year southern Ontario study (Burnett et al., 1995), and are considerd to be due to the limited size of the data set (only 388 days versus 2000+ days in the earlier 1995 study). Due to the high correlations between most of the particle metrics (0.6-0.9 for all except CoH, 0.3-0.5), inter-particle comparisons are doubtful, since the effect of one is likely to be attributed in the regression to another highly correlated metric. Combined regression of the four gaseous air pollutants with and without PM was attempted for both respiratory and cardiac admissions, but the results should be interpreted extremely cautiously in view of the above-mentioned high correlations.

The relationship of hospital admissions for respiratory illnesses to the air pollutants PM_{10}, sulphate, and ozone was examined in Montréal (population almost 3 million) for five summers, from May to October, 1984-1988 (n=911 days) (Delfino et al., 1994). The means and 90th percentile values for PM_{10} and SO_4^{2-} were 29.5 and 45.7 μg/m^3 (PM_{10}) and 4.2 and 8.2 μg/m^3 (SO_4^{2-}), well below international standards for PM_{10} (no objectives exist for sulphate). The 1 h and 8 h ozone values were 36.7 and 60.3 ppb for 1 h O_3 and 30.4 and 51.5 ppb for 8 h O_3, also well below the current Canadian objective of 82 ppb. For five of the six day sampling period for particles, it was necessary to estimate PM_{10}, using the light extinction coefficient, temperature, O_3, NO_x, and barometric pressure. This meant that PM_{10} was actually acting partly as a surrogate for fine particle air pollution from photochemical episodes. The correlation coefficients between PM_{10}, SO_4^{2-}, and ozone were high, as expected, with r=0.90 for SO_4^{2-} and r=0.68 for ozone. The SO_4^{2-}-ozone coefficient was also high, r=0.66. Temperature was also highly correlated with all three of the pollutant variables examined: correlation coefficients were 0.66, 0.58 and 0.72 for PM_{10}, SO_4^{2-}, and ozone. Since the analysis was confined to the warmer season, the variability in the pollution measures aproached a normal distribution, and linear regression methods using Ordinary Least Squares was carried out. A 19 d weighted moving average (Shumway filter) was used to remove longer-wave cyclic yearly and seasonal variations in outcomes (hospitalization counts) and exposures. Temperature, relative humidity and day of the week effects were also controlled. Data were examined for both a longer warm season (May-October) and a shorter summer season (July, August). The effect of high air pollution days on adverse respiratory effects was examined by carrying out the regression after dichotomizing data at days above the 90th percentiles, which were >46 μg/m^3 for PM_{10}, >8.2 μg/m^3 for SO_4^{2-}, and > 60 ppb for ozone (1 h maximum). Pollutants were first examined singly in the models and then other explanatory variables were added. Finally, pollutants which remained significant predictors of hospitalizations were regressed in the same model together.

During May-October, an increase of 10 μg/m^3 PM_{10}, lagged 3 d, was associated with a 2.1% increase in asthma admissions (95% CI 0.6 to 4.0%). After inclusion of ozone, the PM_{10} regression coefficient changed only minimally, but the standard error was markedly inflated resulting in marginal significance, probably due to the high correlation between these two pollutants. For the 46 days in July and August with high air pollution (SO_4^{2-} > 8.2 μg/m^3), non-asthma respiratory admissions associated with sulphate lagged 4 d were 9.6% higher (95% CI 0.5 to 18.7%) than for days that were below 8.2 μg/m^3, the 90th percentile. Asthma admissions and non-respiratory admissions were not affected by SO_4^{2-}.

While ozone (lagged 4 d) was significantly associated with respiratory admissions during July and August when regressed alone, its coefficient dropped by 40% and it lost significance when temperature was included. Temperature itself (lagged 0 and lagged 1 d) was correlated with hospital admission in the all respiratory, respiratory non-asthma and non-respiratory categories, and thus could potentially act as a counfounding factor for ozone, with which it was also highly correlated (r=0.7). Due to the estimation technique for missing PM_{10} values, this study was plagued by the high correlations between the three pollution metrics (PM_{10}, SO_4^{2-}, and O_3), that were examined,as well as the temperature correlations. Therefore, the effects observed on hospital admissions cannot be ascribed to any one component of particulate matter or to particles rather than ozone. The modelled variables should be looked upon as a reasonable expression of the mix of air pollutants affecting respiratory health (Delfino et al., 1994).

Delfino and colleagues attempted to further delineate the effects of the various particle metrics in a smaller study, also in Montréal, in which daily emergency room visits for respiratory illness were examined for the summers of 1992 and 1993 ($n \leq 98$ for each year) (Delfino et al., 1997). There were 10 659 visits for respiratory diseases and over 13 000 visits for non-respiratory causes (the reference group) recorded in 1993. Mean emergency department (ED) visits in 1993 were 98/d for total respiratory diseases, all ages, and 27/d for the elderly. Mean and 90th percentile values for PM_{10} were 22 and 38 μg/m³, and for $PM_{2.5}$, 12 and 22 μg/m³. Mean and 90th percentiles for sulphate were 3.3 and 7.8 μg/m³, for H^+, 4 and 10 nmol/m³, for ozone (8 h), 31 and 46 ppb, and for 1 h maximum ozone, 36 and 57 ppb. As in the previous study, high correlations were observed between most of the air pollutants. Sulphate made up a large fraction of both $PM_{2.5}$ and PM_{10}, and the correlation coefficients were 0.93 and 0.87. Maximum temperature was highly correlated with both measures of ozone (r=0.7), and moderately correlated with the particle measures (r=0.4-0.5). The data were treated as previously by ordinary least squares (OLS) regressions after detrending with a 19 d weighted filter and applying various corrections for season, weather, and day of week effects.

No associations were found for 1992, although both pollutant levels and ED visits were significantly different between years. The authors noted that about one-third of the PM data were lacking for 1992, which could have accounted for the lack of significance in this year, due to increased variance and reduced statistical significance. While no associations were found for most age groups, respiratory visits for the elderly were associated in 1993 with increased O_3 (1 h max. and maximum 8 h moving average), PM_{10}, $PM_{2.5}$, and SO_4^{2-}, all lagged 1 day (corrected for temperature and humidity effects). An increase to the mean level (36 ppb) of (1 h) ozone was associated with a 21% (95% CI 8 to 34%) increase in ED visits. The effects of particulates were smaller with mean increases of 16% (4 to 28%) for 22 μg/m³ PM_{10}, 12% (2 to 21%) for 12 μg/m³ $PM_{2.5}$, and 6% (1 to 12%) for 3.3 μg/m³ SO_4^{2-}. Acidity (H^+) was not associated with ED visits in adults. In pairwise regressions of ozone with PM_{10}, $PM_{2.5}$ and SO_4^{2-}, the regression coefficients for the PM metrics were reduced by half or more, and the the coefficient for ozone was also reduced by about 25%. However, it is not reasonable to assume that the respiratory association with particulate matter was due to ozone, because of the high correlations between PM and ozone (r=0.57-0.63), and it appears more likely that particles and ozone are both independently associated with respiratory ED visits. Tests for statistical interactions between ozone and PM were negative. An isolated effect was found for children under the age of two years with respiratory visits associated with H^+, which the authors suggested was spurious based on internal inconsistencies (Delfino et al., 1997). This study provides limited evidence for an independent association of ozone and three of the four measures of particulate matter with respiratory disease. Acidity did not appear to play a role in exacerbation of disease leading to outpatient visits. The small data sets in this study, combined with the high correlations among all of the air pollutants make it problematic to discern which of the pollutants was the best predictor of adverse respiratory outcomes.

A two-year study of air pollution and respiratory hospital admissions was conducted in the cities of Buffalo, Albany, Metropolitan New York City and suburban N.Y.C. (White Plains) in June to August 1988 and 1989 (Thurston et al., 1992). Air pollution measurements were collected for particulate phase aerosol strong acidity (H^+), sulphate (SO_4^{2-}) and ozone (O_3). Hospital admissions data employed in this study were counts of unscheduled (emergency) hospital admissions with a primary diagnosis of respiratory illness in New York State acute care hospitals in selected counties. The range of mean and maximum SO_4^{2-} values for all areas were 6.9-9.6 μg/m³ and 26.0 to 41.8 μg/m³, respectively. Acidity measurements showed a range of means and maximums from 2.2-3.3 μg/m³ and 7.5-18.7 μg/m³, respectively.

For the summer of 1988 in Buffalo, a 9% increase in respiratory hospital admissions per 10 μg/m³ SO_4^{2-} and a 28% increase per 10 μg/m³ H^+ was calculated from Poisson regression, after adjustment for cyclic and seasonal variations, day of the week effects, and maximum temperature. Ozone was also highly significantly associated. Results for New York City were similar to those for Buffalo. The relative risks for H^+ were consistently higher than those for SO_4^{2-}. Results were not significant for 1989, when pollutant levels were lower than in 1988, nor for Albany or White Plains, N.Y. In the case of Albany, a combination of small population and lower air pollutant values could have been responsible for the lack of association, while in White Plains, the explanation offered was the high socioeconomic status of this population, which tends to mitigate the effects of outdoor pollutants, through use of air-conditioning and remaining indoors during pollution episodes. The results of the present study indicated that summertime haze pollution appeared to play a role in the occurrence of respiratory admissions during that particular season, especially for asthma admissions, for which air pollution was estimated to account for an average of 6-24% of Buffalo and New York City 1988 summertime admissions. At the same time, the relative risk of admissions in these cities increased by a factor of 1.19-1.43 on maximum summertime pollution days in 1988. Because of the high correlation coefficients between ozone and the two particle metrics ($r= 0.51$-H^+; $r= 0.67$-SO_4^{2-}), and between SO_4^{2-} and H^+ ($r=0.77$), the effects of these pollutants cannot be distinguished from one another. The authors considered that the results were consistent with the hypothesis that H^+ acts to exacerbate the effects of ozone on the respiratory disease process (Thurston et al., 1992). Measurements for PM_{10}, and $PM_{2.5}$ were lacking in this study. These PM metrics are usually highly correlated to SO_4^{2-} and H^+. If this were the case in this location, the hospitalization increases noted with SO_4^{2-} and H^+ may well have also been seen with fine particles and PM_{10}.

An extension of the previous study on respiratory admissions in Buffalo, N.Y. has recently been completed (Gwynn et al., 1996). During the summers of 1988 to 1990, PM_{10} (missing days estimated from SO_4^{2-} and CoH data) and three fine particle measurements were investigated (SO_4^{2-}, H^+ and CoH), but their means and ranges were not given. Poisson regression techniques were followed, with filtering to remove cyclic and seasonal variation, and adjustments for day of the week, linear time trends, quadratic hot and cold temperature terms, and a heat and humidity interaction term. For total admissions, a 1 to 2% increase (statistically significant) per equivalent range of $\mu g/m^3$ was calculated (note that means and ranges were not given), for all except CoH, which was only marginally significant. For respiratory admissions, the increases (per the range for each, which was not given) were 20%, 38% and 1-2% (n.s.) for SO_4^{2-}, H^+ and CoH respectively, and for the larger particle size (PM_{10}) the increase was also significant at 18%. All four of the criteria pollutant gases were also significantly correlated with respiratory hospital admissions in single-pollutant regressions ($t= 4$ to 7.5, except for CO, $t= 2$), and they remained significant (NO_2 and CO were marginal) although with a reduced relative risk when co-regressed with acidity (H^+). Acidity was the most consistently associated of the PM metrics when included in models with each of the gases in turn. Based on data presented thus far in this preliminary paper, the order of significance for the four particle metrics was $H^+ > SO_4^{2-} \sim PM_{10} >$ CoH. The particle metrics were said to be all highly correlated with each other, but correlation coefficients were not presented. The high correlations noted would make it impossible to determine the true size of the various PM regression coefficients and thus the metric with the best predictive power for respiratory disease. This paper provided some potentially interesting results but was poorly documented and presented.

Ponce de Leon et al. (1996) investigated the effects of air pollution levels in London, England by recording daily counts of hospital admissions and adjusting for all variables (trend, season and other cyclic factors, day of the week and holidays, influenza epidemics, temperature, humidity, and autocorrelations) in Poisson regressions. Pollution data recorded were Black Smoke (BS), SO_2, O_3, and NO_2, lagged 0-3 d. The study period began in April 1987, ending in February 1992 (1,796 days) and included all ages in three groups, the 0-14, 15-64, and 65+ age groups. Daily average levels of BS and SO_2 were obtained for four monitors, with data for more than 75% of the study days. The reported mean level of BS was 14.6 $\mu g/m^3$ with 5th and 95th percentile values of 6 $\mu g/m^3$ and 27 $\mu g/m^3$, respectively. No relationship between BS and hospital admissions was observed in any age group either for the whole year or for separate analyses of summer and winter. However, ozone was found to be significantly associated with respiratory admissions; an increase of 10 ppb ozone in the warm season was associated with a 1.7% increase (95% CI 0.85-2.5%). The ozone regression coefficient was little changed by addition of BS, NO_2, or SO_2 in bivariate regressions. Although statistically significant health effects in the elderly were associated with NO_2 exposure in the summer, this was attributed to the higher correlation between ozone and NO_2 in summer than in winter or in the entire year. Although some sporadic associations were noted between SO_2 and respiratory admissions in some age groups, the effects were not consistent. The results for particulate matter, despite pollution levels in London within the range associated with effects in other cities, did not appear to be consistent with most other current evidence based mainly on North America. Some explanations were offered for these results including too few monitors (only four) to cover as wide an area as the city of London, and the possibility that BS represented either a less toxic type of particulate than PM_{10} in North America, or BS measurements, which are optically determined based on dark soot particles, may not accurately capture concentrations of the secondarily-formed sulphates and nitrates that are uncoloured. The level of BS in London was one-half or less the values in other European cities in which positive associations were observed.

A daily time-series analysis was conducted on the association between BS, PM_{13}, O_3, SO_2 and NO_2, and hospital admissions for all respiratory diseases, COPD, and asthma in Paris, France for the six-year period 1987-1992 (Dab et al., 1996). Mean BS was 32 µg/m^3; concentrations were higher in winter (mean 40 µg/m^3) than in summer (mean 25 µg/m^3). PM_{13} concentrations were less seasonal, with 51, 54 and 48 µg/m^3 for yearly, winter, and summer means respectively. The increase in all respiratory admissions was 0.41% (95% CI 0.07 to 0.75% for a 10 µg/m^3 increase in BS, and almost the same, 0.45% (95% CI 0.04 to 0.87%) for a similar increase in PM_{13}. Increases were not significantly associated with either BS or PM_{13} for the specific asthma and COPD categories of respiratory disease. SO_2 was associated with all three endpoints (RR for a 10 µg/m^3 increase = 1.10 (24 h) and 1.05 (1 h) for COPD, and 1.07 for asthma), while NO_2, both 24 h and 1 h, was associated with asthma, with the RR for a 100 µg/m^3 increase at 1.18 (95% CI 1.06-1.30) for 24 h NO_2 and 1.08 (95% CI 1.02-1.15) for 1 h NO_2. Ozone was not significantly associated with any endpoint in this analysis, although the RR for a 100 µg/m^3 increase in 1 h ozone was 1.03 (95% CI 0.997-1.073) for all respiratory admissions. No correlations between pollutants were given, and no pollutants were co-regressed (Dab et al., 1996).

In Athens, Greece, during the year 1988, the daily numbers of emergency outpatient visits for all causes, and hospital admissions for cardiac and respiratory causes (diagnoses at time of admission) were examined with respect to BS, CO and NO_2 (Pantazopoulou et al., 1995). Mean resiratory admissions were 41.8/d in winter and 41.4/d in summer (total over 15 000 admissions). Mean cardiac admissions were 73.8/d in winter and 63.9/d in summer (total over 25 000 admissions). Mean emergency visits (all causes) were 592/d (winter) and 575/d (summer). Recorded mean values for six area monitoring locations for BS were 75 µg/m^3 and 55 µg/m^3 for winter and summer, respectively, with 5th and 95th percentile values of 26 µg/m^3 and 161 µg/m^3, and 29 µg/m^3 and 90 µg/m^3, respectively. After adjustment for month of year (5 dummy variables), day of week, temperature (linear) and relative humidity, a positive association was found between the number of visits and air pollution levels for both BS and CO, in winter only. The resulting effects of BS exposure were more pronounced for morbidity of the respiratory system (20-29% increase from 5th to 95th percentile pollution levels) and to a somewhat lesser extent of the circulatory system (15-17% increase from 5th to 95th percentile pollution levels). A 10 µg/m^3 increase in BS was associated with a 1.2% increase in respiratory-related visits and a 0.96% increase in cardiac disease-related visits. Division of the area into three, based on high, medium and low (the above metropolitan area) BS mean concentrations, (wintertime mean daily averages for BS of 135, 112, and 75 µg/m^3) gave poor dose-response information. Results with NO_2 were statistically significant when wintertime NO_2 from the central monitoring station was used as an air pollution indicator. A 10 µg/m^3 increase in NO_2 was associated with a 1.4% increase in respiratory hosital admissions and a 2.0% increase in cardiac admissions in winter. A 10 µg/m^3 increase in CO (in winter) was associated with a 4.0% increase in respiratory admissions and a 2.3% increase in admissions for cardiac disease. The relationship between air pollution and total emergency outpatient visits for all causes (not specifically cardiorespiratory) was also examined but no association was observed with any of the three pollutants. The three measured air pollutants were noted to be highly correlated with each other, but no values were provided. Potential sources of bias in the data were noted such as a lack of a definition of a "real" emergency case, use of admission diagnosis rather than discharge diagnosis, and inclusion of admission cases from outside the Athens area (Pantazopoulou et al., 1995). The high correlations between pollutants precludes assignment of the air pollution effects on cardiorespiratory health to any single pollutant.

As part of the APHEA collaborative European project, Schouten et al. (1996) assessed the short-term relationship between air pollution and the daily number of emergency hospital admissions for respiratory disease in the Netherlands (cities of Amsterdam and Rotterdam) between 1977 and 1989. Pollution parameters used were 24 h daily means for BS, SO_2, and NO_2, one hour maxima for SO_2, NO_2, and O_3 and 8 h maxima for BS, O_3, SO_2, and NO_2. In Amsterdam, black smoke pollution data were available only for the years 1986-1989 and measurements were taken about 20 km west of the city. The annual mean level for BS in Amsterdam was 11μg/m^3 with 5th and 95th percentile values of 1 and 37 μg/m^3. Concentrations were almost twice as high in winter as in summer. In Rotterdam, the annual mean BS level was 26 μg/m^3, with 5th and 95th percentiles of 6 and 61 μg/m^3. Concentrations in Rotterdam were more even throughout the year, with somewhat higher levels in winter (95th percentile values 72 and 54 μg/m^3 in winter and summer respectively). Emergency hospital admissions data were collected for all respiratory admissions (6.7 and 4.8/d in Amsterdam and Rotterdam respectively), chronic obstructive pulmonary disease (1.74/d – Amsterdam and 1.13/d – Rotterdam), and asthma (1.13/d – Amsterdam and 0.53/d – Rotterdam) for all ages, and data were subdivided for adults in the age groups 15-64 years and >65 years. It was observed that the daily counts of emergency hospital admissions due to all diseases of the respiratory system were low in both cities, for all age categories.

Neither BS nor the other pollutants were significantly associated with respiratory disease admissions in Amsterdam, although the RR associated with BS was somewhat elevated for some categories. In Rotterdam, BS (100 μg/m^3 increase) was associated positively but not significantly with all respiratory admissions for all ages (RR 1.24, 95% CI 0.88-1.76), and positively with an increase in RR to 1.37 (95% CI 1.09-1.73) for all respiratory admissions in the age group 15-64 years. The association was significant in summer but not in winter, a finding inconsistent with the somewhat lower concentrations of BS in summer. Ozone was also associated with significant increases in relative risk for all respiratory admissions in the 15-64 and the 65+ age groups (RR for younger adults was 1.15 (95% CI 0.97-1.38) for 8 h ozone averaged over days 0-5; RR for elderly adults aged 65+ in the 1977-1981 period only were 1.25 (95% CI 1.04-1.50) for 8 h ozone and 1.27 (95% CI 1.09-1.49) for 1 h ozone). NO_2 was associated with increased relative risks for all respiratory admissions in the 65+ age category in the 1982-84 period only and for COPD in all age groups together (RR 1.05 to 1.20 for 1 h, daily mean, lagged 2 days or averaged over day 0-2). The low number of admissions, both total and by category, combined with subdivision into age categories, probably contributed to the inconsistent results found in this study. The lower BS concentrations recorded for Amsterdam compared to Rotterdam could also have contributed to the lack of significance in the Amsterdam results. Analysis of the results by summer and winter seasons did not show any consistent pattern. Although some regressions showed higher relative risks in certain categories for the elderly, the pattern was also not consistent. Specificity of effects was also weak, since only NO_2 was associated with COPD, and it was not associated with all respiratory admissions. Analysis of results for the whole Netherlands (population 14 million), in which very little spatial variation in pollutant levels occurred, indicated a relative risk for respiratory admissions, all ages, in the winter season of 1.10 (95% CI 1.024 to 1.184) for an increase of 100 μg/m^3 in BS. The RR was slightly lower, and was not significant in summer, as expected. The relative risks for SO_2, and NO_2 were also significantly elevated in winter, during the period when their concentrations were also highest. The RR for ozone was elevated in summer, when its highest concentrations were recorded. No correlations between pollutants were shown, nor were any bivariate regressions undertaken (Schouten et al., 1996).

BS was again used as the particle metric in Barcelona, Spain, examining the association between the daily number of emergency department (ED) admissions for chronic obstructive pulmonary disease (COPD) and air pollution in the five years between 1985 and 1989 (Sunyer et al., 1993). SO_2 levels were also collected to help determine whether its association with COPD was independent of the role of BS. Summer and winter pollution data were collected separately to help control more adequately for weather and influenza epidemics. Mean BS concentrations were estimated to average 63.2 μg/m^3 over the study period, and were 48 μg/m^3 in summer and 68 μg/m^3 in winter (data from Castellsague 1995). Numbers of daily hospital admissions for Barcelona residents over the age of 14 with COPD were collected and recorded for the study period. In single pollutant analyses, both BS and SO_2 were associated with COPD emergencies in both winter and summer although the association with BS in the summer was not significant. A 2.3% increase (t=5.0) in visits for COPD per 10 µg/m^3 increase in BS and a 2.5% increase per 10 µg/m^3 SO_2 was predicted for winter. In a bivariate analysis, the increases in hospitalizations were reduced but remained significantly different than zero for both pollutants, to 0.94% (t=2.7) with SO_2 and 0.81% for BS (t=2.6) (no confidence limits were provided). The correlations between BS and SO_2 were high,(r=0.7), but the similar reductions for both lend support to the hypothesis of the researchers that both pollutants have independent associations with COPD in Barcelona. Stratification by tertile of each pollutant also showed that both pollutants remained significant, but that there were some inconsistencies. Statistics for other air pollutants were not given. SO_4^{2-} was said to form 38% of BS, but co-regression of SO_4^{2-} with SO_2 did not reduce the SO_2 association. The authors pointed out that BS is not a good measure of particulates in the summer as there is less heating than in winter and therefore less burning of fossil fuels. The ratio of total suspended particulates to BS was also found to be much higher in the winter than in the summer (Sunyer et al., 1993).

Castellsague et al. (1995), also for Barcelona, assessed the relationship between levels of black smoke, SO_2, NO_2, O_3 and adult emergency room visits for asthma during the same five year period from 1985-1989. Analysis was performed separately for the winter (January 1 to March 31) and summer (July 1 to September 30). Mean Black Smoke (BS) was 48 µg/m^3 in summer and 68 µg/m^3 in winter. After adjusting for weather and time-related variables, current day and lagged day 1-3 average summertime BS were associated with a 3.3% (95% CI 0.4-6.3%) and a 4.6% (increase, respectively, per 10 µg/m^3 increase in BS. The increase was small and was not significant in winter. NO_2 was associated with small but significant increases in asthma in both summer (1.8%; 95% CI 0.4-3.2%) and winter (2.2%; 95% CI 0.4-4.2%), while SO_2 and ozone were not significant in this analysis in either season. Because of high collinearity between variables, no co-regressions were undertaken. Therefore it is not possible to ascertain the independent relationship of BS with emergency hospital visits for asthma in this study.

Walters et al. (1994) examined the relationship between BS and SO_2 with hospital admissions for asthma and all respiratory diseases in Birmingham, UK for the two years April 1988-March 1990. No measurements of NO_2, O_3 or other pollutant gases were available. The mean daily BS level was low, at 12.7 µg/m^3 (max. 188), and mean daily (24 h) SO_2 was 39 µg/m^3 (max. 126), averaged for seven sites. Both SO_2 and BS peaked in concentration from October to December. The relationships were examined seasonally, by stepwise Least Squares regression, after inclusion of temperature, humidity and temperature. Since admissions lagged behind peaks in pollution, lags of 0 and 2 d, and the 0 to 3 d moving average lag were examined. BS was found to be more closely association with all respiratory disease admissions than with asthma admissions, and was also more strongly associated than SO_2. Associations were significant for the winter season, and to a limited extent in the summer (although it was pointed out by the authors that this could be the result of confounding with unmeasured pollutants including ozone and pollens). In winter, 25% and 20.5% increases in respiratory admissions, lagged 2 d, were associated with increases of 10 µg/m^3 BS and SO_2 respectively. Increases of 21% and 17% in asthma admissions, also lagged 2 d, were associated with BS and SO_2 respectively (per 10 µg/m^3). Acidity did not appear to play a role in exacerbation of disease leading to patient visits. Co-pollutant regression revealed that BS, but not SO_2, was the independent determinant of increased respiratory and asthma admissions (complete data not presented) (Walters et al., 1994). The authors did not comment on the lack of correlation between autumn admissions and the highest yearly pollutant levels at that time.

12.3.4 Summary of Acute Hospitalization and Emergency Department Studies

These studies cover a wide range of circumstances surrounding exposure to particulate matter. Summaries of the findings for PM_{10} and BS are presented in Tables 12.3 and 12.4. Results for $PM_{2.5}$ are shown in Table 12.5, while results for sulphate are shown in Table 12.8.

Of the 16 studies that examined PM_{10} and respiratory endpoints (Table 12.3), all showed associations of PM_{10} in univariate analyses with one or more respiratory endpoints, at means between 25 and 55 µg/m^3. The total respiratory hospitalizations category was considered in eight studies. The relative risk, as a percentage increase per 10 µg/m^3, varied from 0.45% (Paris, France) to 4.7% (Toronto, Thurston et al., 1994) with a median of 1.7 µg/m^3 , and was higher for emergency department visits, at 7.3% in Montréal.

Black Smoke (BS), a particle metric that is intermediate in size between PM_{10} and $PM_{2.5}$, has been used extensively in Europe. In six of eight studies (Table 12.4), the relative risk for respiratory hospitalizations varied widely from 0.4% (95% CI 0.07 to 0.75%) to 12.3% (95% CI 5.8-18.2%) per 10 μg/m^3 increase in BS. The range of means in positive studies was from 12.7 to 75 μg/m^3. The different airborne concentrations recorded in the various cities, as well as the relative importance of other co-occurring air pollutants, could have accounted for some of these differences. In Amsterdam, NL, no air pollutants were found to be associated with hospitalizations, possibly a reflection of low numbers of hospitalizations and consequent low power of the study to detect such effects, while in London, UK, only ozone (not BS, NO_2, or SO_2) was consistently associated with increased respiratory hospitalizations. In both cities, ambient BS concentrations were quite low, less than 15 μg/m^3 versus higher than 26 μg/m^3 for all of the other locations except Birmingham, in which only BS and SO_2 were measured and BS was associated in bivariate regressions with increased hospitalizations.

Overall, there is some evidence for an association of BS with increases in respiratory disease, despite the variations observed in the strength of the association and in its statistical significance, and the lack of data on its significance when included with the range of gaseous pollutants known to occur with it. BS is an optical measurement of dark-coloured particles, predominantly from power generation, heating fuel and diesel combustion. It does not measure uncoloured particles such as sulphates or nitrates secondarily formed in the atmosphere after primary combustion. It may not, therefore, be a particularly good indicator of the type of particulate matter air pollution which has been prevalent since the 1980s in urban areas, and which is due predominantly to vehicular fuel combustion (Anderson et al., 1996; Walters et al., 1994).

Only three hospitalization studies, two in Toronto and one in Montréal, directly examined the association between fine particles as $PM_{2.5}$ and respiratory or cardiac effects. Positive associations between $PM_{2.5}$ and respiratory effects (2.5 to 9.6% per 10 μg/m^3) were seen in univariate analyses in all three studies (Table 12.5). The association was slightly reduced from 2.5 to 1.8% in Toronto (Burnett et al., 1997) when ozone was included in the analysis, but was no longer significant in the earlier Toronto study (Thurston et al., 1994) or in Montreal (Delfino et al., 1996) after inclusion of ozone. The high correlation between ozone and $PM_{2.5}$ in these two studies (r=0.7 – Thurston et al. (1994) and r=0.46 to 0.63 – Delfino et al. (1996)) makes it difficult to separate out the individual effects of $PM_{2.5}$ from ozone. However, in the larger Toronto study by Burnett et al. (1997) in which the correlation coefficent was lower (r<0.34), the $PM_{2.5}$ association remained after including ozone. The Burnett et al. (1996) study also examined cardiac effects, and found that the relative risk was not significantly elevated in association with $PM_{2.5}$, regressed either singly or with ozone. Additional information on the magnitude of the risk of respiratory hospitalizations associated with $PM_{2.5}$ is given by the large study on sulphate and ozone in southern Ontario (Burnett et al., 1995) (Table 12.5). An overall site-specific conversion factor for sulphate to $PM_{2.5}$ was determined (see Appendix; $PM_{2.5}$ =6.973897 + 1.917519 SO_4^{2-}), and indicated that the relative risk for respiratory causes (adjusted for ozone, temperature, etc.) of 3.5% (95% CI 2.3-4.7%) for a 13 μg/m^3 increase in sulphate was equivalent to a 31.9 μg/m^3 increase in $PM_{2.5}$, or 1.1% (95% CI 0.7 to 1.5%) for a 10 μg/m^3 increase in $PM_{2.5}$, which was slightly lower than the above previous estimates, but within their confidence intervals.

All eight studies (Table 12.8) which examined SO_4^{2-} found positive associations with respiratory endpoints (2 to 9%, and up to 18.2% in an emergency department study), at mean concentrations between 3.3 (Montréal) and 11.9 μg/m^3 (Toronto, summer 1988). The association became marginal or disappeared in the two Toronto studies and in the Montréal ED visit study when ozone was included in the regression with SO_4^{2-}. However, the association remained significant but somewhat reduced in strength in the ozone-adjusted regressions in the two southern Ontario studies, which were by far the largest and most reliable of those available. The correlations between ozone and SO_4^{2-} were high in all studies, 0.5 to 0.8, which causes difficulties in separating out the effects of one from the other. Overall, SO_4^{2-} has good evidence for an association with respiratory hospitalizations and is considered to be a good surrogate for fine particles from combustion sources. Cardiac effects were significantly associated with SO_4^{2-} in the southern Ontario study, with an ozone-adjusted increase of 2.5% (95% CI 1.3-3.7%) in cardiac-related hospitalizations for a 10 μg/m^3 increase in SO_4^{2-} (data analyzed over the whole year) (Burnett et al., 1995). In a second, smaller study on data for three summers in Toronto, a 4.4% increase per 10 μg/m^3 increase in sulphate was observed in the univariate analysis, but was no longer seen in the bivariate analysis with ozone (Burnett et al., 1997).

Table 12.3 Summary of Findings on Hospital Admissions and Emergency Department Visits Associated with PM_{10}

Location Reference	PM_{10} mean, (range) $\mu g/m^3$	Endpoint	% increase per 10 $\mu g/m^3$	Other pollutants considered
Utah Valley +Cache, Salt Lake Valleys Pope et al., 1991	53, 55, <40 (3 locations) (max 365)	respiratory, all ages (total 4144 admissions) pneumonia bronchitis asthma	Rates doubled in Utah Valley compared to Salt Lake or Cache Valley when steel mill open or when $PM_{10}>50$ 1.7% (RR=1.17) 4.0% (RR=1.40) 0% (RR=1.14, not significant)	No other pollutants considered (SO_2 was low, near detection limit)
Seattle, WA Schwartz et al., 1993	29.6 (6 -103)	(ED visits, all ages) -asthma (7.1/d)	3.7% (1.2 to 6.4%) (<65 y) (n. signif., >65 y)	SO_2, O_3 not significant (RR = 0.99 for SO_2) RR= 0.97 for O_3) β_{ext} also assoc, RR=1.11 (1.04-1.18) Main source of PM was wood smoke
Birmingham, AB Schwartz 1994a	45 (19, 77) (10th, 90th percentile)	(>65 y) -pneumonia (5.9/d) -COPD (2.2/d)	1.9% (0.7 to 3.2%) 2.7% (0.8 to 5.0%)	O_3 (24 h) pneum RR=1.14 (0.94-1.38) O_3 (1 h) COPD RR=1.07 (0.96-1.20) No bivariate regression. (PM_{10}-O_3 correlation =0.29)
Detroit, MI Schwartz 1994b	48 (22, 90) (10th, 90th percentile)	(>65 y) -pneumonia(15.7/d) -COPD (5.8/d)	1.2% (0.4 to 1.9%) (+O_3) 2.0% (0.9 to 3.2%) (+O_3)	O_3 (24 h) positive; Pneumonia RR=1.052, COPD RR=1.054 (per 10 ppb) Bivariate regression did not alter coefficients for PM_{10} or O_3, i.e. both independently associated. (PM_{10}-O_3 correlation =0.35)
Detroit, MI Schwartz & Morris 1994	48 (22, 90) (10th, 90th percentile)	cardiovascular, >65 y -ischemic heart disease (44/d) -congestive heart failure (27/d)	0.56% (0.2 to 1.0%) CHF not significant.	PM_{10} RR 0.75% (0.13-1.4%) + CO. O_3 not sign. CO RR=1.22 (1.01-1.03) but n.s. in bivariate regr. SO_2 RR=1.014 (1.003-1.026 for IQ range) but n.s. + PM_{10} (i.e. CO, SO_2 not independent of PM_{10}.)
Minneapolis, MN Schwartz 1994c	36 (18, 58) (10th, 90th percentile)	(>65 y) -pneumonia (6/d) -COPD (2/d)	1.7% (0.3 to 3.3%) 5.7% (2.0 to 10.6%)	PM_{10} RR same or higher + O_3 O_3 indep. assoc. (RR=1.15) with pneum. in co-regression with PM_{10} (i.e. both independently assoc.)
New Haven, CT Schwartz 1995	41 (67, 90th percentile)	(>65 y) respiratory (8.1/d)	1.2% (0.0 to 2.6%; p<0.05)	PM_{10} + O_3: PM 1.8%, O_3 1.4% (10 ppb). PM+ SO_2: PM_{10} same, SO_2 0.8% PM_{10} and O_3 indep. assoc., SO_2 probably not (SO_2 n.s. with O_3)

Table 12.3 Summary of Findings on Hospital Admissions and Emergency Department Visits Associated with PM_{10} *(continued)*

Location Reference	PM_{10} mean, (range) µg/m^3	Endpoint	% increase per 10 µg/m^3	Other pollutants considered
Tacoma, WA Schwartz 1995	37 (67, 90th percentile)	(>65 y) respiratory (4.2/d)	2.0% (0.6 to 3.4%)	PM_{10} + O_3: PM n.s. ($p>0.1$), O_3 RR same PM_{10} + SO_2: PM_{10} RR same, SO_2 n.s. (RR=0.99). Ozone dominates over PM_{10} and SO_2
Spokane, WA Schwartz 1996	46 (83, 90th percentile)	all respiratory (>65 y) (3.9/d) -Pneumonia (1.9/d) -COPD (1.0/d)	1.7% (0.7 to 2.7%) 1.1% (-0.03 to 2.5%) 3.4% (1.6 to 5.4%)	SO_2 negligible in Spokane. 1-h O_3 also assoc., 9.5% for 10 ppb. Not co-regr. because only 115 days measured together.
Anchorage, AK Gordian et al., 1996	45.5 (5-565)	(Doctors= visits) -asthma, -upper respir. tract infections	3.5% ($p<0.01$) 1.2% ($p<0.05$)	CO assoc.with URI, bronchitis. Benzene high. Limited data for other pollutants. O_3 max=40 ppb (2y). SO_2, NO_2 low.
E. Washington State Heflin et al., 1994	40 (3-1700)	-bronchitis -asthma	0.35% ($p=0.03$) not significant	No other pollutants considered Dust storms resulted in increased otitis, UR infections.
Toronto, Ont. Thurston et al., 1994	30,30,39 (3 yrs.)	-respiratory -asthma	4.7% ($p=0.015$) 0.44% (not significant)	O_3 also associated in bivariate regression with all PM metrics. H^+ remained significant, SO_4^{2-} marginally so.
Toronto, Ont. Burnett et al., 1997	25 (10-58)	-respiratory -cardiac	2.5% ($t=3.42$) 2.3% ($t=2.24$)	PM_{10} coefficient retained value when regressed with all gases except NO_2 (but r high). Strength of association: CoH $>PM_{2.5}>SO_4^{2-}>PM_{10}>H^+>$CP. O_3 was indep. associated in bivariate regressions with all PM
Buffalo, NY Gwynn et al., 1996	not given	-respiratory	-18% (12 - 24% for range)	$H^+>SO_4^{2-}=PM_{10}>$CoH
Montréal, Qué. Delfino et al., 1994	31.5 (July-Aug)	-asthma -respir. non-asthma	2.1% (0.6 to 4.0%) 1.0% ($t=1.83$; n.s.)	PM_{10} RR for asthma reduced to 1.8% (marg. sign) in bivariate regression with O_3 and O_3 also reduced (n.s.), but r=0.63 SO_4^{2-} included
Montréal, Qué. Delfino et al., 1996	30.1, 21.7 (2 y)	(Emerg. Dept.visits) -respiratory	7.3% (1.95 to 12.7%)	PM_{10} reduced to 4.5% (n.s.) with O_3. O_3 significant with all PM (H^+, SO_4^{2-}, $PM_{2.5}$, PM_{10}). Limited evidence for independent associations of O_3 and 3 of 4 PM, but PM-O_3 and O_3 - temperature correlations high.
Paris, France Dab et al., 1996	50.8 (max 138) (PM_{13})	-all respiratory -COPD -asthma	0.45% (0.04 to 0.87%) -0.5% not significant -0.025% not significant	SO_2 significant, all 3 endpoints NO_2 significant for asthma BS ~ PM_{13}

Table 12.4 Summary of Findings on Hospital Admissions and Emergency Department Visits Associated with Black Smoke (BS)

Location	Reference	BS mean, (range) µg/m³	Endpoint	% increase per 10 µg/m³	other pollutants considered
London, England	Ponce de Leon et al., 1996	14.6 (6-27)	-respiratory disease	-2 to +2% (n.s.)	O_3 RR unchanged (independent) in bivariate regr. with BS. Correlation = 0.3 to 0.45). NO_2, SO_2 n.s.
Birmingham, Eng.	Walters et al., 1994	12.7 (IQR 5-15; max 188) 12.3 (winter)	-respiratory dis. -asthma	12.3% (5.8-18.2%) – winter 2.8% (0.3-5.3%) – winter	BS+SO_2: BS sign., resp.+ asthma, SO_2 neg. Alone, SO_2 sign, resp; marg asthma
Amsterdam, NL	Schouten et al., 1996	11 (1-37)	-respiratory dis. -COPD -asthma	1.3% (-3.1 to 8.7%- 65+) 4.7% (-1.9 to 16.5%) -2.0% (-5.6 to 4.8%)	No correlations or co-regressions. O_3 , SO_2 and NO_2 also not signif.
Rotterdam, NL	Schouten et al., 1996	26 (6-61)	-respiratory dis. -COPD -asthma	3.7% (0.9 to 7.3%) 2.4% (-1.2 to 7.6%) n.s. (Not given)	No correlations or co-regressions. O_3 marg, resp (adults), NO_2 sign., COPD (all ages). SO_2 not signif.
Paris, France	Dab et al., 1996	31.9 (max 268) 40 – winter; 25 – summer	-respiratory -COPD -asthma	0.41% (0.07 to 0.75%) -0.05% (n.s.) 0.43% (n.s.)	No correlations or co-regres. PM_{13} similar to BS. O_3 n.s.; SO_2 +ve all endpts. NO_2 +ve, asthma.
Athens, Greece	Pantazopoulou et al., 1995	75 – winter 55 – summer	(Emerg Dept visits) -respiratory dis. -cardiac	1.2% (winter) 0.96% (winter)	High collinearity (values not given). No co-regressions. CO, NO_2 also significant in winter.
Barcelona, Spain	Sunyer et al., 1993	68 (IQR 45-84) winter 48 (IQR 31-63) summer	(Emerg Dept visits) -COPD	2.28% (t=5.0) (winter) 0.94% (t=2.7) + SO_2	In bivariate regr., both BS and SO_2 coeff. reduced, but BS remained signif. in winter. In summer, SO_2 sign., BS not. BS – SO_2 r = 0.7 (high)
Barcelona Spain	Castellsague et al., 1995	68 (IQR 45-84) winter 48 (IQR 31-63) summer	(Emerg Dept visits) -asthma	0.9% (-0.8 to 2.6%) winter 3.3% (lag 0 d) summer 4.6% (lag 0-3 d) summer	No co-regression. BS r=0.32 to 0.73 for O_3, NO_2, SO_2. NO_2 sign., but O_3, SO_2 not signif.

Table 12.5 Summary of Findings on Hospital Admissions and Emergency Department Visits Associated with $PM_{2.5}$

Location **Reference**	**$PM_{2.5}$ mean (range), µg/m³**	**Endpoint**	**% increase per 10 µg/m³ (95% confidence limits)**	**Effect of other pollutants considered**
Toronto, Ont. Thurston et al., 1994	18.6 (3 y, summer) (17.7, 15.8, 22.3)	respiratory asthma	9.3% (1.3-17.3%; p=0.01) 3.7% (-1.6-9.3%; p=0.08)	$PM_{2.5}$ associated in single regression, but not in bivariate regression with O_3. SO_4^{2-}-O_3 correlation coefficient high, r=0.7. Other PM also n.s. + O_3
Toronto, Ont. Burnett et al., 1996	16.8 (IQR 8-23) (3 y, summer)	respiratory cardiac	2.5% (t=3.3, significant) 2.1% (t=1.8, not significant)	$PM_{2.5}$ +O_3: 1.8% (t=2.3, significant, 28% lower) $PM_{2.5}$ +O_3: 0.9% (t=0.8, not signif., 55% lower)
Montréal, Québec Delfino et al., 1996	18.5 (90th percentile 44) 12.2 (90th percentile 22) (Summers, 1992,93)	respiratory (emergency dept., age >64 years)	9.6% (1.9 to 17.3%) (1993) (1992 results not associated)	$PM_{2.5}$ +O_3: 4.5% (not significant, but $PM_{2.5}$-O_3 correlation coefficient very high, r=0.62) PM and O_3 likely independently associated.
Southern Ont. Burnett et al., 1995	SO_4: 4.37 (95th percentile 13) [$PM_{2.5}$: 15.3 (95th percentile 31.9) – based on site-specific conversion factor from SO_4^{2-} to $PM_{2.5}$]	respiratory asthma COPD infections cardiac coronary disease dysrhythmias heart failure	[1.1% (0.7-1.4%)] [1.0% (0.4-1.8%)] [1.5% (0.7-2.2%)] [1.0% (0.5-1.5%)] [1.0% (0.5-1.5%)] [0.7% (0.2-1.5%)] [0.4% (-0.6-1.4%)] [0.9% (0.2-1.7%)] [all results based on site-specific SO_4^{2-} – $PM_{2.5}$ conversion]	Figures included adjustment for ozone. Results of single pollutant regression virtually the same as results for bivariate regression with ozone. PM and ozone independently associated.

No evidence for a threshold of effects for respiratory hospitalizations associated with particulate matter or other air pollutants was found in nonparametric concentration-response curves (in which a linear or other defined response is not assumed), or in quartile or decile plots by concentration, at the low (10 to 100 μg/m^3 PM_{10}) concentration ranges examined. Curves appeared to increase monotonically, with steep slopes at low concentrations and some suggestion of curvilinear responses (lower slope) at higher concentrations. Nonparametric concentration-response curves were shown for PM_{10} and hospitalizations due to pneumonia and COPD in the elderly in Birmingham, AB (Schwartz 1994), and for respiratory disease hospitalization in Toronto, ON associated with PM_{10}, coarse particles between 2.5 and 10 μm, $PM_{2.5}$, SO_4^{2-}, CoH and H^+ (Burnett et al., 1997).The curves were stated to be not significantly different from linear in the Toronto study, although a pronounced plateau between 19 and 35 μg/m^3 was observed for fine particles ($PM_{2.5}$), and the response appeared to be somewhat curvilinear for coarse particles and SO_4^{2-}. A decile plot for SO_4^{2-} and respiratory hospitalizations in southern Ontario showed a curvilinear response, with a steep slope from 0-6 μg/m^3, and a less steep slope between 6 and 20 μg/m^3 (Burnett et al., 1994).

Quartile analyses were undertaken in Seattle, WA for PM_{10} and asthma (Schwartz et al., 1993), Detroit, MI for PM_{10} and respiratory or cardiac hospitalizations (Schwartz 1994b; Schwartz and Morris 1995), southern Ontario for SO_4^{2-} and respiratory or cardiac hospitalizations (Burnett et al. 1995), and Barcelona, Spain for BS and COPD or asthma (Sunyer et al., 1993; Castellsague et al., 1995). In general, the respiratory responses were shown to increase monotonically with increasing concentration, despite the loss of data due to aggregation of responses into only four concentration points for each endpoint and location. The concentration-response for cardiovascular disease in the elderly in Detroit was clearly curvilinear (Schwartz and Morris 1995). The curve for COPD admissions in Detroit was the only one showing an anomalous response, since hospitalizations for quartile two (at 30 μg/m^3) were higher than for quartile three (at 50 μg/m^3). The quartile analyses were also used to determine a "Lowest-Observed-Adverse-Effect Level (LOAEL)" for respiratory hospitalizations around 20 to 30 μg/m^3 with a "best estimate" of 25 μg/m^3 for PM_{10}.

The specificity of the air pollution effect on the causes of disease was investigated by considering potential associations with cardiovascular disease as well as respiratory disease. A stronger association with air pollution was observed for respiratory diseases than for cardiovascular diseases, in most of the regressions from the four studies which examined cardiovascular diseases in addition to respiratory disease. In southern Ontario, the relative risk (RR) for cardiovascular diseases was 2.5% (95% CI 1.3 to 3.9%) for a 10 μg/m^3 increase in SO_4^{2-}, compared to 2.7% (95% CI 1.9 to 3.6%) for respiratory disease (Burnett et al., 1995). This stronger association with respiratory disease hospitalizations was also evident for SO_4^{2-} and fine particles ($PM_{2.5}$) in a much smaller study carried out in Toronto, where the risks for cardiovascular diseases hospitalizations were lower by up to one-half the RR for respiratory disease and were not significant (Burnett et al., 1997). However, the RR estimated for cardiovascular diseases using other particle metrics, PM_{10}, CP (particles between 2.5 and 10 μm), H^+ and coefficient of haze (CoH), either remained the same or were higher than those for respiratory disease. Similarly, in Detroit, MI, Schwartz (1994) and Schwartz and Morris (1995) found higher relative risks (per 10 μg/m^3 increase in PM_{10}) for respiratory disease admissions (2.0% for COPD and 1.2% for pneumonia) than for ischemic heart disease (0.56%) or heart failure (0.75%). In Athens, Greece, a 10 μg/m^3 increase in BS was associated with an 11.7% increase in respiratory hospitalizations and a smaller increase of 9.6% for cardiac hospitalizations. The RR for CO and NO_2, which were also associated with increased hospitalizations in this city, were one-half to two thirds as high for cardiac as for respiratory disease admissions (Pantazopoulou et al., 1995).

Some of the larger studies were able to examine the respiratory subcategories chronic obstructive pulmonary disease (COPD), asthma, and/or pneumonia or infectious diseases. Individuals with pre-existing COPD have been suggested as particularly susceptible to the effects of air pollution. The Burnett et al. (1995) study on the entire age range in the population of southern Ontario indicated that the increased hospitalization risk for COPD was somewhat higher than that for total respiratory admissions, approximately 3.7% per 10 μg/m^3 increase, using SO_4^{2-} as the particle metric. The European studies with BS as the particle metric did not find an association with increased hospitalizations for COPD, except for the Sunyer et al. (1993) study in Barcelona in which both BS and SO_2 were implicated. The association between PM_{10} and COPD was strong, 2.4 to 5.7% per 10 μg/m^3 increase, in a series of studies on the elderly in four US cities (Schwartz 1994a-c, 1996). However, in these latter studies the authors did not examine and compare total respiratory admissions or any other age category.

A number of investigators examined the association between asthma and particulate matter, with mixed results. The hospitalization data provide only limited evidence for an association between particulate matter and asthma. The best evidence is provided by the southern Ontario Study, which had the most extensive database (42 asthma admissions/day over six years) (Burnett et al., 1994, 1995). While asthma showed an association with increased daily airborne sulphate concentrations, the association in the year-round analysis was about the same or slighly lower in magnitude than that for general respiratory admissions, 3.2% versus 3.5% for a 10 $\mu g/m^3$ increase in sulphate (Burnett et al., 1995). In the May-August period, the asthma rate was slightly higher, 7.1% versus 5.8% for the total respiratory admission rate (based on increases in a combination of 50 ppb ozone and 5.13 $\mu g/m^3$ SO_4) (Burnett et al., 1994). The asthma rate was highest (13%) for infants ≤ 1 year, and was also high (8.2%) for children <14 years old, and for middle-aged adults (9.8%) between 35 and 64 years. Asthma admission, doctors' visits or emergency department visits were also associated with PM_{10} increases in Montréal, Qué. (2.1%), Anchorage, AK (3.5%) and Seattle, WA (4.0%) (all per 10 $\mu g/m^3$ increase) (Delfino et al., 1994; Gordian et al., 1996; Schwartz et al., 1993). The positive association in Seattle was seen only for those less than 65 years old, and not in the elderly. In Montréal, sulphate (the only fine particle measured) showed no association (Delfino et al., 1994), while in Seattle, the PM_{10} was largely attributable to wood smoke particles in the fine fraction (Schwartz et al., 1993), and therefore the absent association between hospitalizations, doctors' visits and emergency department visits, and PM_{10} could be at least partially a fine particle effect. In univariate analysis, the association was marginal between asthma and SO_4^{2-} in Buffalo, NY (Thurston et al., 1992). In a bivariate analysis with ozone, $PM_{2.5}$ was associated marginally (3.7%; p=0.08) with asthma in Toronto, ON, while the associations with PM_{10} and SO_4^{2-} were negative (Thurston et al., 1994). PM_{10} was not associated with asthma in eastern Washington State (Hefflin et al., 1994) or in Detroit (the elderly, who appear to be less affected by asthma than the general population, were the only group included) (Schwartz 1994b). In univariate analyses, BS was not associated with asthma in Paris, France, Amsterdam and Rotterdam NL (Dab et al., 1996; Schouten et al., 1996), but a positive association was seen in Birmingham, UK, which disappeared when BS was regressed together with SO_2^{2-}, also associcated with asthma in Birmingham (Walters et al., 1994). The small number of respondents in many of these studies (e.g., 0.75/d in Detroit) could have been part of the reason for some of the lack of positive associations with respect to asthma. The size of the particle or its chemistry did not appear to play a role in determining the outcome. The southern Ontario study, with the clearest positive results for asthma, also included exposure to ozone, which has also been linked to asthma. This suggests the possibility that particulate matter might work in concert with ozone or another co-occurring air pollutant in the exacerbation of underlying asthma. It also suggests a relatively small independent role for particulate matter air pollution in exacerbation of asthma.

Laboratory studies indicating that exposure to high concentrations of airborne particles may act to compromise the immune system, have suggested that epidemiology studies should investigate the exacerbation of respiratory infections as one of their endpoints. In the southern Ontario study conducted by Burnett et al., (1994) (one of the few studies large enough to be able to subdivide data on the basis of endpoint and age without losing the power of the study to detect effects), increases in overall infection-related hospitalization rates were associated with daily increases in SO_4^{2-} and ozone combined, but the overall rate increase (4.3%) was lower than the total respiratory rate increase (5.8%), and was driven almost entirely by the infant infection-related rate (19.1%). The 1995 analysis of these data (Burnett et al., 1995) more clearly separated out the effects of SO_4^{2-} on infection rates, while retaining adjustments of the presence of ozone. The infection rate attributable to a 10 $\mu g/m^3$ increase in SO_4^{2-} was 2.5% (95% CI 1.3 to 3.7%), about the same as the asthma rate increase and slightly lower than the overall infection rate increase of 2.7% (no breakdown of age versus infection rates was presented) (Burnett et al., 1995). Two other, much smaller, studies in Utah and Anchorage, AK also recorded increases in hospitalizations due to pneumonia or upper respiratory tract infections of about 1.2% per 10 $\mu g/m^3$ increase in PM_{10}. Increases of 1.1 to 1.9% in hospitalizations for pneumonia were observed in a series of studies with the elderly ($\geq$ 65 years) in four US cities (Schwartz et al., 1994a-c, 1996). The epidemiology studies on hospitalizations thus support the conclusion that particulate matter acts to exacerbate infections, but suggest that this effect is most marked in infants, and that it is not as important a source of increases in hospitalizations in the population at large as other pre-existing conditions.

There are some concerns as to the reliability of some of the results from hospital admissions data owing to potential confounding. The coding of the different sub-categories of respiratory disease and the differentiation between some categories of cardiac and respiratory admissions by use of International Classification of Disease (ICD) codes which may differ between hospitals or individual doctors, may cause some misclassifications. However, it should be noted that misdiagnosis does not fluctuate in concert with pollution levels and therefore will not affect the overall conclusions with respect to pollutant concentrations, but may affect the conclusions with respect to which categories of disease are most influenced by pollutants. The possibility that certain epidemics of contagious respiratory disease can occur coincidentally with particulate pollution episodes was also considered by some investigators.

The effects of age on hospitalizations or emergency department visits were examined in several locations, since historical data from episodes of high air pollution strongly suggested that it was the elderly, the young, and those with pre-existing respiratory and/or cardiovascular conditions who were responding to air pollution. While some studies (Delfino et al., 1996; Burnett et al., 1995; Schouten et al., 1996) found that the elderly were at increased risk compared to other age groups in the population, the increases observed in cardiorespiratory hospitalizations were by no means predominantly due to effects on the elderly. In the Burnett et al. (1994) study in southern Ontario, warm season sulphate and ozone air pollution was associated with smaller increases in respiratory hospitalizations in the elderly (4.3% for 50 ppb ozone and 5.1μg/m^3 SO_4^{2-} increases) than in those between age 2 and 64 years (5.5% for age 2-34 years and 7.2% for those aged 35-64 years). In a later expanded analysis of the same database, Burnett et al. (1995) reported similar increases (per 13 μg/m^3 increase in SO_4^{2-}, after taking ozone into consideration) in respiratory hospitalizations of 3.7% and 3.8% for younger adults (age 15-64 y) and the elderly (65+ y), which were higher than those seen in children (2.7% for children under 15 years), but the effects of age were evident in the data for cardiovascular hospitalizations, in which a 3.5% increase (95% CI 1.9-5.0%) was seen in the elderly compared to a 2.5% increase (95% CI 0.5-4.8%) for those less than 65 years of age. In Rotterdam, NL, the 15-64 year age group showed the strongest association between BS and all respiratory admissions, with a RR of 1.37 (95% CI 1.09-1.73) compared to a RR of 0.97 (not significantly different from 1.0) for those aged 65 or older (Schouten et al., 1996). However, for other air pollutants NO_2 and SO_2, the elderly appeared to be at greatest risk. In Amsterdam in the same study, the elderly appeared to be at greatest risk for respiratory disease associated with BS and all other included air pollutants (O_3, SO_2, and NO_2), although small numbers in subdivided groups meant that most risks were not significantly elevated (Schouten et al., 1996). In London, UK there were no consistent differences in relative risk of respiratory hospitalizations for those over age 65 compared to those between 15 and 64 y (Ponce de Leon et al., 1996). With asthma as the endpoint, the elderly were at less risk than those under age 65 years, in Seattle, WA (Schwartz et al., 1993), Anchorage AK (Gordian et al., 1996) and southern Ontario (Burnett et al., 1994). The series of studies by Schwartz (1994a-d, 1995) did demonstrate that the elderly were affected by air pollution and that elevated respiratory hospitalization rates were associated with increases in particulate air pollution, but only the elderly were studied in these cities due to limitation of the hospitalization database to this age group only.

Children were also shown to be a high-risk group for increased respiratory disease in a few studies. These included the Pope (1991) study on the Utah Valley comparing effects of respiratory hospitalizations when a steel mill (the predominant source of particulate air pollution in this area) was open as when it was closed. Bronchitis and asthma admissions were twice as high for preschool children (ages 1-5 years). By comparison, for all ages combined, the rates were only 1.4 times as high for bronchitis and 1.2 times as high for pneumonia (open mill verus closed mill). In Anchorage, AK, upper respiratory tract infections were most strongly associated with PM_{10} in children less than 10 years old and in older adults aged 45 years or more, compared to adolescents and younger adults (Gordian et al., 1996). While summertime ozone and sulphate combined appeared to have a greater effect on increasing respiratory hospitalizations in infants and children (14.8%) than on adults (4.3-7.2%) in southern Ontario (Burnett et al., 1994), all-year sulphate was associated with lower respiratory hospitalizations in children (2.7%; 95%

CI 0.8-4.7%) than in adults (3.7-3.8% for 13 μg/m^3 increases in SO_4^{2-}) after accounting for the effects of ozone (Burnett et al., 1995). The London, UK respiratory hospitalization study (Ponce de Leon et al., 1996) did not find that children were at increased risk for respiratory admissions due to BS or other air pollutants, at least not at the concentrations experienced in the 1980s and early 1990s.

Most of the studies handled potential confounding by temperature and season well, using a variety of methods, but there has been concern that the effects observed are due to the rise and fall of other unmeasured pollutants in concert with PM. In the Utah studies, most of the potential confounders were accounted for or ruled out. The studies by Schwartz et al., (1993) showed strong associations after taking factors such as weather conditions, seasonal variations and outbreak of infectious diseases into account, but not always after accounting for pollutant covariates.

Most of the hospitalization studies on particulate matter looked at at least one or two other air pollutants as potential covariates; although two (Pope et al., 1991; Hefflin et al., 1994) considered only PM_{10}.

The relationship of SO_2 to hospitalizations was examined in Detroit, MI, New Haven, CT, Tacoma, WA, Toronto, ON (two studies), and in all seven European cities (it was determined to be virtually absent in one location, Spokane, WA). In univariate regressions, SO_2 was associated with all respiratory admissions In Birmingham, UK (Walter et al., 1994), Paris, France (Dab et al., 1996), New Haven, CT and Tacoma, WA (Schwartz 1995),and in Toronto, ON (Burnett et al., 1997), with COPD in Paris (Dab et al., 1996) and Barcelona (Sunyer et al., 1993), and with asthma in Paris (Dab et al., 1996) and Birmingham, UK (Walters et al., 1996). SO_2 was also found to be asociated with cardiovascular disease in Detroit and Toronto, in univariate analysis (Schwartz and Morris 1995; Burnett et al., 1997). No associations were found with respiratory disease (also in univariate analyses) in London, UK, Amsterdam or Rotterdam, NL, Barcelona (for asthma only), Seattle, WA or Toronto, ON (Schwartz et al., 1993; Thurston et al., 1994; Castellsague et al., 1995; Ponce de Leon et al., 1996; Schouten et al., 1996).

However, in the six studies that undertook bivariate analyses with both PM and SO_2 in the regression model, SO_2 was not clearly shown to have an association that was independent of particulate matter air pollutants. In New Haven, CT, the regression coefficient for SO_2 was slightly increased when it was included with PM_{10}, but was reduced, and no longer significant, when included with ozone. This effect was even more marked in Tacoma, WA, with the RR for SO_2 declining from 1.06 in univariate regression to 0.99 with PM_{10} and 0.93 wih ozone (no correlation coefficients between these pollutants were given) (Schwartz 1995). While the bivariate analysis in Barcelona indicated that both SO_2 and BS were associated with COPD independently of each other based on similar drops in regression coefficients (with retention of statistical significance for both), the stratification by tertiles of each pollutant tested against the other indicated that BS, but not SO_2, was independently associated with COPD (Sunyer et al., 1993). In the Birmingham, UK case, BS, but not SO_2, was associated with all respiratory hospitalizations and with asthma visits in winter, in stepwise linear regressions with both pollutants incuded in the same model (Walters et al., 1994). In Toronto, Burnett et al. (1997) regressed SO_2 with each of six particle metrics in turn. For respiratory admissions, the RR for all particle metrics was significantly elevated, while the RR for SO_2 (adjusted for temperature and dew point) was reduced by about one-third, from 1.033 to 1.022, still significant or marginally so for all particle metrics except CoH and PM_{10}. For cardiac admissions, SO_2 remained significant with H^+, SO_4, and $PM_{2.5}$ (none except H^+ was significant in univariate analysis). In Detroit, MI, the relative risk for ischemic heart disease was reduced from 1.014 to 1.009 (95% CI 0.99 to 1.023) in bivariate regression with PM_{10}, while the PM_{10} RR was reduced only by about one-sixth. This indicated that SO_2 was not independently associated with cardiac hospitalizations but that PM_{10} was the best air pollution metric of those that were examined, including PM_{10}, ozone, CO, and SO_2 (Schwartz and Morris 1995).

NO_2 was examined for possible associations with respiratory disease in eight cities: London, UK, Amsterdam and Rotterdam, NL, Paris, France, Barcelona, Spain, and Athens, Greece, as well as Toronto, ON and Buffalo, NY. Correlations between NO_2 and PM were generally high, and for this reason few investigators were able to perform bivariate regressions including NO_2 with other air pollutants. NO_2 was associated with total respiratory admissions in Athens (1.4% for a 10 μg/m^3 increase), Paris (0.4%), Toronto, ON (0.44%) and Buffalo NY (approximately 0.5%) (Pantazopoulou et al., 1995; Dab et al., 1996; Burnett et al., 1997; Gwynn et al., 1996). NO_2 was also associated with COPD in Rotterdam

(2.0%; 95% CI 0.8 to 4.3% for 1 h maximum, with a cumulative lag period of 0 to 3 d), but in this city, BS was not associated with COPD. NO_2 was also associated with asthma in Paris (RR=1.175; 95% CI 1.06-1.30 for a 100 µg/m^3 increase), and in this case also, BS was not a significant predictor of asthma (Dab et al., 1996). In Barcelona, 1 h NO_2 was associated with asthma in summer with a RR of 1.8% (95% CI 0.36 to 3.24% for 10 µg/m^3 increase) and in winter (RR=2.2%; 95% CI 0.4 to 4.2%), while BS was also associated in summer (RR=3.3% (0.4 to 6.3%) but not in winter (RR=0.93%) (Castellsague et al., 1995). In London, UK, the authors of the study (Ponce de Leon et al., 1996) considered that NO_2 was not associated with respiratory admissions because the association did not appear to be robust (inconsistencies were evident in regressions categorized by age and by season), but it may have been dismissed without sufficient reason. It was associated with admissions in both the warm season and all-year regressions for all ages, and in the warm season for children, and all-year for the elderly. However its regression coefficient went down by one-third in bivariate regressions with ozone, which was the air pollutant best expressing effects on respiratory health in this city, while BS was not associated with respiratory hospitalizations (the ozone-NO_2 correlation was moderately low at 0.29 in summer, and -0.43 in winter, allowing the testing of effects with both pollutants included). In North America, Gwinn et al. (1996) found NO_2 to be associated with respiratory hospitalizations in Buffalo, NY (RR= approximately 0.5% for an IQR increase which was not given), and the association was retained when regressed together with particle acidity (H^+). Burnett et al.(1997) also found associations between NO_2 and respiratory hospitalization both singly (RR=4.4% for a 6 ppb or IQR increase), and in bivariate analyses with all six PM metrics tested (RR= 1.033-1.037 or 3.3 to 3.7%). However correlations between NO_2 and the particle metrics were high (0.45-0.61 for $PM_{2.5}$, PM_{10}, the coarse fraction between 2.5 and 10 µm, and CoH) or moderate for SO_4^{2-} (0.34) and moderately low for H^+ (0.25), which calls for the questioning of the results for the high group.

Cardiac admissions were associated with increased NO_2 in Athens (2.0% in a univariate regression; Pantazopoulou et al., 1995)) and in Toronto in a univariate analysis(0.5% for an IQR increase of 6 ppb). This association remained significant in bivariate regressions with H^+ (RR=0.38%; H^+ remained significant), SO_4^{2-}, PM_{10}, $PM_{2.5}$, and the coarse fraction between 2.5 and 10 µm, (0.39%, 0.36%, 0.38%, 0.23% respectively, with non-significant RR for all 4 particle metrics) but not with CoH (RR=-0.1%; CoH RR=0.7%). Overall, it was not possible to distinguish the effect of NO_2 from particles or other air pollutants due to high correlation coefficients between them, but there was suggestive evidence that the role of NO_2 in respiratory and/or cardiac illnesses requires further investigation.

The possibility that an association exists between CO and hospitalizations was examined in only one study for respiratory admissions in Athens Greece (Pantazopoulou et al. 1995) and in the same study, as well as in a study in Detroit MI (Schwartz and Morris 1995) for cardiovascular (CV) disease. In Athens, CO was more strongly associated with respiratory than with cardiac admissions in univariate regressions (26.5% versus 15.3% for a 6.6 µg/m^3 increase in CO). No bivariate regressions or other estimates of the potential independence of the association were undertaken, and the authors noted that the CO-PM correlation coefficient was very high. In Detroit, CO was found to have a strong association with increased risk for congestive heart failure (RR=1.02; 95% CI 1.01-1.03) which was unchanged in bivariate regressions with PM_{10} (Schwartz and Morris 1995). PM_{10} was also shown to be independently associated, although its relative risk fell by about 25% in the bivariate analysis (RR=1.024; 95% CI 1.004-1.044 in bivariate analysis, versus RR=1.032 in single analysis). The relatively low correlation between CO and PM_{10} in this location supports the independent role of both pollutants in exacerbation of cardiovascular diseases.

Ozone in addition to particulate matter was examined in most locations for possible associations with respiratory hospitalizations. In single regressions, after adjustment for weather, season, year and day of the week effects, an association was found between ozone and respiratory hospitalizations in 12 locations (but marginally in three of these). The relative risk for a 10 ppb increase in ozone varied from 1.012 (southern Ontario) to 1.098 (Spokane, WA) with a median of 1.026 (Burnett et al., 1994; Schwartz 1996). In three locations (Seattle, Birmingham, AB, Paris, France), no association was found between ozone and respiratory admissions when ozone was regressed singly. There was also no association between increased ozone and cardiovascular disease in Detroit MI (Schwartz and Morris 1995). In bivariate regressions with particulate matter in nine locations (5- PM_{10}, 3- SO_4^{2-}, 2- $PM_{2.5}$ with overlap), ozone retained its association with respiratory hospitalizations in all nine locations, and its regression coefficient and relative risk was relatively unchanged or even greater than in the single regressions. Particulate matter was also associated in addition to ozone in all cases except Montréal, in which $PM_{2.5}$ was no longer significant in the bivariate regression with ozone, and in London, UK, in which BS was not significantly associated in either single or bivariate regressions. This association with respiratory hospitalizations was strongest for PM_{10} in three locations (Detroit, MI, Minneapolis, MN, New Haven, CT) and for ozone in the other six locations. In the bivariate regressions with ozone, SO_4^{2-} remained associated in Toronto (Thurston et al., 1994; Burnett et al., 1997) and southern Ontario (Burnett et al., 1994), as did $PM_{2.5}$ in Toronto, both with reduced regression coefficients. In a further study in Toronto, ozone was tested against six particulate matter metrics, all of which remained associated independently of ozone except SO_4^{2-}, for which the correlation coefficient with ozone was higher (0.53) than the other particulate matter metrics (0.20-0.34 for $PM_{2.5}$, PM_{10}, coarse particles between 2.5 and 10 µm, particle strong acidity, or CoH) (Burnett et al., 1997). Thus, the evidence is considered to point to the independent association of both particulate matter and ozone with respiratory hospitalizations.

Thus, of the five different air pollutants considered in single and multiple analyses in various locations, particulate matter was the air pollutant with the most consistent and stable association with increases in hospitalizations. Ozone was also shown to be associated independently of PM in some analyses, in six locations with a greater magnitude of association than PM itself. Indications that CO was also associated with cardiorespiratory hospitalizations were observed in two studies, and this finding deserves to be followed up. SO_2 was not clearly shown to have an association independent of particulate matter in locations where bivariate regressions could be undertaken. While NO_2 was found to be associated with respiratory hospitalizations in several univariate analyses, its independence from PM could not be properly evaluated because of the high correlation between NO_2 and PM in most or all locations.

12.4 ACUTE EFFECTS – RESPIRATORY HEALTH

A number of studies have examined the effects of short-term variations in particulate pollution on pulmonary function and/or respiratory health, including measures such as absences from school or work due to respiratory illnesses. Both cross-sectional and time-series methods of analyzing data were used in these studies. The geographical areas range greatly, as do the sources of the particulates. Most of the studies have been done on panels of children.

Some concern has been expressed that the evaluation of respiratory symptoms is too subjective, but these studies provide valuable information on adverse health effects less severe than those requiring hospitalization or resulting in premature mortality, and they potentially affect many more individuals in the population than the more severe effects. In addition, most studies used well standardized and validated methods for determining adverse respiratory effects. The significance of slight changes in pulmonary function has also been questioned, but these tests are objective; moreover they have been shown to be predictive of future respiratory health status.

12.4.1 Acute Effects – Lung Function and Respiratory Symptoms

Studies on PM_{10}

Pope et al. (1991) conducted a study evaluating respiratory health and PM_{10} in the Utah Valley in the winter of 1989-1990. The principal source of PM_{10} in the study area was an integrated steel mill built during World War II. Respiratory symptoms (trouble breathing, runny or stuffy nose, wet or dry cough, wheezing, fever, rash, burning, aching or red eyes, upset stomach) and peak expiratory flow (PEF) measurements were recorded daily over the winter of 1989-90 in 55 participants (34 school children aged 9-11 years with respiratory symptoms, based on questionnaire responses, and 21 asthma patients aged 8-72 years). During the study period, PM_{10} levels ranged from 11 to 195 µg/m^3, with a mean of 46 µg/m^3. Limited monitoring conducted for SO_2, NO_2 and O_3 showed that concentrations of all were considerably lower than the then-current standards; NO_2 averaged 0.03 ppm (annual standard 0.05 ppm), mean SO_2 was <0.01 ppm (24 h standard 0.14 ppm) and ozone was virtually absent in wintertime in Utah. Strong particle acidity never reached the detection limit of 8 nmol/m^3. An increase in PM_{10} of 150 µg/m^3 was associated with a 3 to 6% decline in peak expiratory flow rate (PEF) in both groups. The current-day level had the largest effect, but effects were noted for up to three days later. Results from the regression analysis indicated that a 10 µg/m^3 increase in PM_{10}

was associated with a decline of 0.66 L/min in PEF rate. The mean percentage reporting symptoms of respiratory disease also increased with increasing PM_{10} pollution in the school-based sample, but not in the asthma patient sample. In the schoolchildren, the estimated probabilities of reported symptoms of upper and lower respiratory disease were found to be 1.5 and 2.1 times higher when 24 h PM_{10} levels were at the highest level (195 $\mu g/m^3$) compared with when levels were at their lowest level (11 $\mu g/m^3$). The odds ratio for lower respiratory tract symptoms was 1.25 (95% CI 1.06-1.55) for a 50 $\mu g/m^3$ increase in PM_{10}, and for upper respiratory tract symptoms, it was 1.18 (95% CI 1.03-1.33). Extra medication use increased by two to three times in both groups as PM_{10} levels rose from less than 50 $\mu g/m^3$ to 100 or more $\mu g/m^3$. This study provides some evidence that the impacts of PM_{10} on children with pre-existing respiratory symptoms, (a potentially susceptible subgroup) may be more severe than for a randomly-selected group of children, since comparison of an earlier study showed that 74% of the Utah children had negative responses to air pollution, compared to 59% in a similar study in Steubenville, Ohio (Dockery et al., 1982, in Pope et al., 1991). The lower associations between PM_{10} and both PEF and respiratory symptoms in the asthma patient-based sample may not imply that asthmatics are less susceptible to the effects of air pollution, but may indicate that the asthmatic subjects were managing their symptoms with medication.

Pope and Dockery (1992) expanded the above study in the winter of the following year (100 days from December 6, 1990 to March 15, 1991), examining the association between daily changes in respiratory health and PM_{10} pollution in 39 symptomatic and 40 asymptomatic fifth- and sixth-grade children selected from the same population as previously. As previously, other air pollutant concentrations were very low. Peak flow measurements were recorded by the participants three times a day, and the following respiratory symptoms were reported in a daily diary: trouble breathing, runny or stuffy nose, wet cough, dry cough, wheezing, fever, rash, burning, aching, red eyes or upset stomach. During the study period, 24 h PM_{10} levels averaged 76 $\mu g/m^3$ and ranged from a minimum of 7 $\mu g/m^3$ to a maximum of 251 $\mu g/m^3$. Several pollution episodes occurred during the study period, during which the 24 h US national standard of 150 $\mu g/m^3$ was exceeded up to 14 times. A negative association was found between peak flow measurements and PM_{10} pollution levels in both groups, a 10 $\mu g/m^3$ increase being associated with a PEF decrease of 0.36 L/min in the symptomatic children and 0.26 L/min in the asymptomatic children. The mean PEF of the symptomatic children was also 11 L/min (3.6%) lower than the asymptomatic children, which indicates that this group is not only more affected by PM_{10} increases, but also starts with a slightly compromised lung function. Five-day moving-average PM_{10} levels were more strongly associated with peak flow than were concurrent-day PM_{10} levels, suggesting that response and recovery may last several days and that the timing of response and recovery varies between children. The PM_{10} data were divided into quartiles, with means of 25, 55, 89, and 141 $\mu g/m^3$, and the changes in PEF showed a good dose-response for both groups that was not significantly different from linear. The mean percentage of children reporting symptoms of respiratory disease increased with PM_{10} pollution. Symptomatic children reported higher upper respiratory symptoms (57%), lower respiratory symptoms (82%) and cough (100%), during days when PM_{10} levels were in the highest quartiles than during days when PM_{10} levels were in the lowest quartile. Although no association between pollution and upper respiratory symptoms was found in asymptomatic children, there was approximately a 40% and 78% increase in lower respiratory symptoms and cough during days with pollution levels in the highest quartile compared with days with pollution levels in the lowest quartile, respectively (Pope and Dockery 1992).

Again, as indicated in the previous study, the researchers identified a sensitive subgroup, symptomatic children. The positive results observed in the asymptomatic children indicate that PM_{10} pollution could potentially affect a wide range of children, giving rise to transient changes in pulmonary health (Pope and Dockery, 1992).

Children were again subjects of a study examining respiratory symptoms through a diary in the Gardanne coal basin, near Marseille, France (Charpin et al., 1988). The study was conducted between December 1983 and March 1984 using 450 schoolchildren in the third, fourth and fifth grades. Health effects were evaluated before and after an extension of the power plant was implemented. Pollution data were recorded for respirable particles and SO_2. No size range was given for what was included in respirable particles; it could be assumed that this represents either PM_{10} or PM_{15}. Monthly means ranged from 46 to 92 $\mu g/m^3$, with a minimum and a maximum of 21 μg/m^3 and 262 μg/m^3, respectively, for the four-month study period. The questionnaire obtained information on symptoms such as morning cough, eye irritation, runny nose, sneezing, sore throat and wheezing. Researchers found no correlation between daily prevalence of symptoms and daily respirable particle levels. Smoking in the home, a common potential confounder in this type of study, was not mentioned and therefore probably not controlled for (Charpin et al., 1988).

Associations between respiratory function in children and heavy industrial pollution were examined in Cubatao, Brazil, during the 1988 school year (March-June, August-November) (Spektor et al., 1991). Six hundred six-year-old children, attending six different kindergarten schools, were tested monthly for standard spirometry measures. Pollution in the area was primarily of industrial origin: iron and steel mills, fertilizer, cement and gypsum production and coke kilns, as well as chemical, paint and many other secondary plants. In addition, the city is located on the coast and surrounded by mountains, which leads to frequent trapping of air pollutants. Measurements recorded included PM_{10}, SO_2 and O_3 levels, as well as $PM_{2.0}$ and coarse particles, as PM_{10-2}, (these were not further considered in this preliminary analysis). Yearly average PM_{10} concentrations ranged from 59 μg/m^3 to 110 μg/m^3 in the six school areas (with children participating in the study living within 500 metres of the schools). Lung function data were regressed against mean monthly PM_{10} levels, and significant associations were found with reductions in FEV_1 (-0.3 to -0.6 ml/μg/m^3), PEF (-1.2 to -2.6 ml/sec/μg/m^3), and FEF_{25-75} (-1.1 to -3.1 ml/sec/μg/m^3) for the month prior to measurement. The reporting of pollution data for the six school sites was inexact. However, this study area does appear to be an ideal area for future studies because of the wide range of pollution levels and sources in the different regions of the valley (Spektor et al., 1991).

Studies with various measures of fine particles:

Perry et al. (1983) studied the effects of particulate air pollution in adult asthmatics as measured by PEF, symptoms of airway obstruction and aerosolized bronchodilator use. The study was conducted in Denver, Colorado, from January through March 1979, during which time measurements were recorded for both fine (<2.5 μm) and coarse (2 μm to 15 μm) particulates, SO_4^{2-}, NO_3, SO_2, CO and O_3. No relationship was found between most of the air pollution variables measured and peak expiratory flow, symptoms of exacerbation of asthma, or medication use. Fine sulphates had a borderline effect on increases in medication use ($p=0.065$), and fine nitrates were associated with increased symptoms ($p=0.02$) and increased medications both in the morning ($p=0.04$) and evening ($p=0.02$). Pollutant levels were not reported, making it difficult to interpret the data. It was also mentioned that there were very few days of high pollution during the study period.

A daily air monitoring program was conducted in Denver, Colorado, between November 1987 and February 1988 (Ostro et al., 1991), in which 207 asthmatics between the ages of 18 and 70 years were asked to provide daily diary information on asthma symptoms, medication use, utilization of medical services, indoor exposures as well as other variables related to measured pollutants in the area. The pollutants measured twice daily included $PM_{2.5}$, sulphates, nitrates, acidity and SO_2. Mean, minimum and maximum levels of $PM_{2.5}$ were reported as 22, 0.5 and 73 μg/m^3, respectively. $PM_{2.5}$ concentrations were only mildly associated with overall asthma ratings ($p<0.1$), but not with indicators of respiratory health such as cough or shortness of breath. Acidity, as hydrogen ion, was associated marginally with asthma exacerbations, and with significant increases in cough and shortness of breath ($p<0.05$). These effects were more marked after adjustment for time spent indoors. Sulphate was associated with symptoms of shortness of breath. The strengths of this study were good reporting of pollution measurements and screening of potential study candidates. However, the selection of all participants from a single clinic, a majority of whom were white females, employed and well educated, with an average age of 46, formed a sample that is clearly not representative of the general population, thus limiting the validity of the results with respect to the general population (Ostro et al., 1991).

A panel study with 155 asthmatic children and 102 asthmatic adults was recently carried out over two winters in three neighbouring cities, Erfurt and Weimar in former East Germany, and Sokolov, Czech Republic (Peters et al., 1996a). Possible associations were investigated between the air pollutants SO_2, TSP, PM_{10}, SO_4^{2-}, particle strong acidity, and the health indicators of respiratory symptoms and lung function as peak expiratory flow rate (PEF). Children's PEF was significantly reduced by increases in the 5 d means of all the pollutants. SO_4^{2-} and SO_2 provided the strongest reductions (1.1 and 0.9%

respectively), for their interquartile ranges (t=4.0 for both). The fine particle measures were more strongly associated with these reductions than the particle measures PM_{10} or TSP, which both included coarser fractions. For example, the PEF reduction associated with SO_4^{2-} (interquartile range) was twice as great as that with particle strong acidity, while it was 2.6 and 3.2 times greater than the reductions associated with PM_{10} and TSP. A 15% increase in adverse respiratory symptoms was significantly associated only with the gaseous pollutant SO_2 for an interquartile range increase of 128 µg/m^3 in the 5 d mean. Concurrent day air pollution was generally not associated with any endpoints, except for a 0.43% decrease in PEF associated with PM_{10} (52 µg/m^3 interquartile range), about the same magnitude as the decrease for the 5 d mean (48 µg/m^3 range). On the basis of a 10 µg/m^3 increase in pollution for each of the particulate pollutant metrics, the percentage decreases in PEF were 1.2% for SO_4^{2-}, 0.14% for PSA, 0.09% for PM_{10} and 0.05% for TSP (Peters et al., 1996a).

The results for the adults in the same study are as follows. In adults, effects on PEF were smaller and less consistent; a 0.4% decrement in PEF was significantly associated only with 5 d average TSP (interquartile range increase of 71 µg/m^3), equivalent to 0.06% for a 10 µg/m^3 increase. Symptom responses were more marked in adults than in children. Five-day average SO_2, PM_{10} and PSA were all significantly associated with symptom increases (18%, 15%, and 11% for respective interquartile range increases), while TSP and SO_4^{2-} were not. Concurrent day pollutant indices were not associated with any endpoints in adults. Thus, in this study, asthmatic children were found to be more responsive than asthmatic adults in the same communities, but less responsive regarding symptoms. It was noted that medication use was frequent in both children and adults, which could have modified effects differentially among people with pre-existing respiratory conditions (Peters et al., 1996a). This study, although small and with some monitoring data gaps, was carefully conducted. It also gave indication that the fine fraction $\leq$2.5 µm, as SO_4^{2-} or H^+ (PSA), had a greater effect on children's PEF (but not on symptoms) than other larger particle measures. However, the results in this regard were inconsistent for adults. The authors did not co-regress SO_2 with any of the particle measures. TSP and PM_{10} were highly correlated (r=0.63-0.88), and PSA was moderately correlated (r=0.43) to the other particle measures. About 15% of PM_{10} was composed of SO_4^{2-}.

Peters et al. (1996b) investigated the relevance of numbers of fine particles compared to their mass, during the winter 1991-92 (145 days), in Erfurt, former East Germany. A mobile opticle particle counter and a differential mobility analyzer gave number and mass concentrations of particles less than or equal to 2.5 µm in diameter. Seventy-nine percent of the particles were smaller than 0.1 µm in diameter, while 82% of the mass concentration was associated with particles in the size range 0.1 to 0.5 µm. The evening mean PEF of a panel of 27 non-smoking asthmatic adults (44-80 years old) were regressed against the 5 d mean (lag 0-4 d) of number counts and equivalent particle mass concentrations, after adjustments for weekends, 24 h mean temperature, and a linear trend. While declines in PEF for the interquartile ranges of all measured fractions were significant, the decline in PEF was more pronounced for the number concentrations than for the equivalent mass concentrations. An interquartile range (IQR) increase in number concentration (PM<0.1 µm) of 9200/cm^3 (79% of all particles) was associated with a decline of 4.04 L/min (95% CI -2.0 to -6.1), while an IQR mass concentration ($PM_{0.1-0.5}$) of 28.8 µg/m^3 (82% of all mass) was associated with a smaller decline of 2.1 L/min. (95% CI -0.6 to -3.7) (p=0.04). Similarly the IQR for the number concentration of the entire $PM_{2.5}$ sample, 9950/cm^3, was associated with a decline of 3.8 L/min (95% CI -2.0 to -5.6), while a smaller decline of 2.3 L/min (95% CI -0.5 to -4.1) was associated with the IQR of 36.0 µg/m^3 for the $PM_{2.5}$ mass concentration (Peters et al., 1996b). This is perhaps the first epidemiology study which has attempted to examine the role of the submicron fraction of particulate matter in respiratory function. Although a very small study, requiring confirmation in other work on larger populations and on other endpoints, it provides supporting human data for the animal toxicology work which has suggested that ultrafine particles are the most important fraction with respect to cardiorespiratory toxicity.

Dassen et al. (1986) measured the effect of exposure to $PM_{2.5}$ during an air pollution episode on the pulmonary function of 163 primary schoolchildren in the Ijmond area, The Netherlands. From January to February 1985, mean levels of respirable suspended particulate (RSP) ($\leq$3.5 µm in size) remained well below 100 µg/m^3, whereas maxima reached 200-250 µg/m^3 during a five-day pollution episode. Measurements were represented by 24 h averages taken once every three days. Other pollutants measured were TSP and SO_2. The source of the particles was generally wind-blown iron ore, coal sand and vehicle exhaust from a nearby industrial area. Researchers recorded five parameters of pulmonary function, including FVC, $FEV_{0.5}$ (forced expiratory volume in 0.5 seconds), FEV_1, PEF (peak expiratory flow), MMEF (maximum mid-expiratory flow), $MEF_{25\%}$, $MEF_{50\%}$ and $MEF_{75\%}$ (maximum flow at 25, 50 or 75% of vital capacity). The periods of high pollution (200-250 $\mu g/m^3$ over five days) for all three pollutants were associated with a transient decline in pulmonary function (percentage not specified). This decline appears to have persisted for at least two weeks, but not longer than 3.5-4 weeks after the episode. The authors suggest that effects are dependent on the duration of the polluting episode. A two-day increase in 24 h average levels of all three pollutants (RSP, TSP and SO_2) in the range of 100-150 $\mu g/m^3$ was not associated with a decline in pulmonary function, suggesting that the episode was not long enough to cause changes in pulmonary function lasting more than one day (Dassen et al., 1986).

In a study by Hoek et al. (1990), pulmonary function was measured in 630 primary schoolchildren living in the Ijmond region, near Amsterdam, The Netherlands. In January 1985, an air pollution episode occurred during which levels of respirable suspended particulates (< 3.5 µm), TSP and SO_2 were recorded in the range of 200-250 $\mu g/m^3$ (24 h averages). Pulmonary function (FVC, FEV_1, PEF, MMEF) was measured during the episode as well as two weeks and 3.5 weeks after the episode. These data were compared with baseline measurements taken when pollution levels were much lower. Most lung function variables were significantly decreased during and, to a lesser extent, two weeks after the episode. There were no significant differences from baseline levels in observations made 3.5 weeks after the episode. Lack of detailed pollution levels and short study duration make it difficult to interpret these data.

Another study by some of the same researchers was conducted involving 112 children, 7-12 years of age, in Wageningen, a small non-industrial town in the eastern part of The Netherlands (Hoek and Brunekreef 1993). Pulmonary testing (FVC, FEV_1, PEF) as well as respiratory symptoms (throat irritation, cough, cough with phlegm, wheeze, runny/stuffed nose, aching throat, shortness of breath, chest tightness, eye irritation, sneezing) were matched against measured levels of PM_{10}, SO_2, NO_2 and black smoke (BS). PM_{10} was measured by daily 24 h measurements. No average PM_{10} level was reported for the study period, but monitoring data did appear to show that the range of PM_{10} levels was ~10-174 $\mu g/m^3$. Results showed that during a winter episode of moderately elevated concentrations of PM_{10}, SO_2 and Black Smoke, pulmonary function of the schoolchildren was significantly lowered. There was no association between the prevalence of respiratory symptoms and air pollution concentrations. It was calculated that FVC, FEV_1 and MMEF decreased by ~2.5% when same-day PM_{10} levels increased by 100 $\mu g/m^3$. It was also pointed out that the magnitude of the response would be considered small (Hoek and Brunekreef, 1993).

Between December 1990 and March 1991, acute respiratory effects as measured by PEF were studied in 73 children aged 6-12 with chronic respiratory symptoms in two small non-industrial towns, Wageningen and Bennekom, The Netherlands (Roemer et al., 1993). Children were screened through a questionnaire, and 74 participants were chosen from 131 available children. No major sources of pollution exist in either of the towns or in their vicinities, thus implying that pollution levels are mainly due to long-range transport. Ambient concentrations of PM_{10}, SO_2, NO_2 and BS were recorded. Maximum, minimum and mean PM_{10} levels were moderate, at approximately 180, 15 and 55 $\mu g/m^3$, respectively. In addition to peak flow measurements, participants were asked to register the occurrence of acute respiratory symptoms (cough, phlegm, runny or stuffed nose, shortness of breath, etc.) and medication use. Small but statistically significant associations were observed between moderately high levels of PM_{10} and decreases in peak flow. Daily prevalence of wheeze and bronchodilator use also had a positive correlation with PM_{10} levels. Other respiratory symptoms were also associated with PM_{10}, but only in a weak positive correlation. Reporting of pollution levels in this study was not clear, and therefore rough estimates were calculated from the data. It is also difficult to determine which pollutant was responsible for the effects observed, as almost all of them were equally associated with the measured health effects (Roemer et al., 1993).

Another study was conducted by Hoek and Brunekreef (1994) on the respiratory health of Dutch children over three consecutive winters (1987-1988, 1988-1989 and 1989-1990) in three non-industrial commmunities and one non-industrial city in the Netherlands. Pulmonary function testing (between 6 and 10 manoeuvres) was performed on more than

1000 children (546 male, 532 female) between the ages of 7 and 11. The occurrence of acute respiratory symptoms (hoarseness, cough, cough with phlegm, wheeze, runny/stuffed nose, aching throat, shortness of breath, chest tightness, eye irritation and sneezing) was registered by the parents in a diary. Information on ambient levels of PM_{10}, fine particle sulphate, fine particle nitrate and nitrous acid was collected. No separation of fine and coarse particles could be accomplished with the sampler used. The 24 h mean PM_{10} level for the study was 44.9 $\mu g/m^3$, with a minimum and a maximum of 14.1 and 126.1 $\mu g/m^3$, respectively. A weak negative association was found between lung function and PM_{10}, as well as most of the other air pollution measurements. Daily prevalences and incidences of symptoms were not clearly associated with any of the air pollutants measured in this study.

Two studies (acute and chronic) were conducted in cities in Montana in the 1978-1979 and 1979-1980 school years (Johnson et al., 1990). The acute study in 1978-79 consisted of a comparison of schoolchildren's (8-10 years) lung function as a function of air pollution levels in one community. Approximately 375 third- and fourth-grade children in the town of Missoula were tested. Measurements were conducted for both TSP and respirable particles (coarse and fine) every three days. Coarse particles included those with aerodynamic equivalent diameters greater than 2.5 µm and less than 15 µm, and fine particles included those less than or equal to 2.5 µm. The sources of the particulates were identified as wood smoke and entrained dust. Results for fine and coarse particulates (24 h sampling) were grouped into categories of 0-30 $\mu g/m^3$, 31-60 $\mu g/m^3$ and >60 $\mu g/m^3$ and results for TSP were grouped into categories of 0-100, 101-150, and 151-200 $\mu g/m^3$. Pulmonary function measurements recorded were FVC, $FEV_{0.75}$ (forced expiratory volume in 0.75 seconds), FEV_1, FEF_{25-75} (mean forced expiratory flow during the middle half of FVC) and PEF. Fine particles showed a trend toward association with small concentration-dependent declines in lung function of 0.65% to 0.75% for FVC (p=0.04-0.08) and 0.47 to 0.5% for FEV_1 (not significant) for the highest category, 61+ $\mu g/m^3$, compared to the lowest category, 0-30 $\mu g/m^3$. The coarse fraction between 2.5 and 15 microns in size was not consistently associated with any lung function endpoint. An increase from the first to the third category of TSP was marginally associated with a decline of 1.0% in FEV_1 (p=0.07). This was confirmed in a followup test program the next year, in which an average decline of 1.65% in FEV_1 was associated with a rise from the winter average of 119 $\mu g/m^3$ to 440 $\mu g/m^3$ in a pollution episode. Declines were also noted in FVC and FEF_{25-75}, but were significant only in females. Thus, fine particles appeared to affect the overall pulmonary volume (FVC), whereas the larger particles affected pulmonary rate (FEV_1), supporting the theory that smaller particles are able to penetrate deeper into the human pulmonary system.

Respiratory health was measured in 2199 schoolchildren in the community of Port Alberni, located at the head of an inlet on the west coast of Vancouver Island (Vedal et al., 1991). The primary sources of pollution in the area are pulp mill boilers and residential wood-burning. The site is bounded on one side by high mountains and is often subject to temperature inversions, which contribute to air pollution problems. In a pilot study in 1986, it was determined that reduced sulphur compounds, SO_2 and methyl mercaptan levels were low in the area. Particulate matter monitoring was conducted between 1989 and 1990, during which TSP was measured for 24 hours every six days at 13 sites. Two sets of rotating monitors were also used to measure PM_{10}, changing sites every six days. From these data, PM_{10} values were predicted for all the sites. A health questionnaire was administered, and spirometry was performed measuring forced expiratory parameters. Two sites had 24 h PM_{10} concentrations that exceeded 150 $\mu g/m^3$. This occurred 2% of the time. Eleven of the 13 sites had 24 h PM_{10} concentrations that exceeded 50 $\mu g/m^3$, occurring 32% and 21% of the time. Estimated PM_{10} and submicrometre particle concentrations were associated with increased chronic cough, chronic phlegm production, congestion, wheezing symptoms and hospitalization for a chest illness before age 2. Threshold concentrations were observed at 35-40 $\mu g/m^3$ for PM_{10} and 20 $\mu g/m^3$ for submicrometre particles. Asthma prevalence was not associated with any of the particle measurements, nor was there an association found between particle exposure and changes in lung function measurement (Vedal et al., 1991).

Respiratory health, measured as the incidence of cough, lower respiratory symptoms and upper respiratory symptoms, was studied in a randomly selected cohort of 1844 schoolchildren in six US cities or approximately 300 children per city (Schwartz et al., 1994). A daily diary of respiratory symptoms was kept by the parents of each child for a period of a year, beginning in 1984. Each home was also visited, and socioeconomic status, home characteristics, and parental smoking were recorded. Community measures for daily PM_{10}, $PM_{2.5}$, $PM_{2.5}$ sulphur (as a measure for sulphate), and acidity (H^+) were recorded, as well as the gaseous air pollutants ozone, SO_2, and NO_2, along with temperature. For the non-winter months of April to August (chosen because ozone was relatively high at this time and the incidence of infectious diseases, which could act as a confounder, was low) the median and maximum PM_{10} (combined for the six cities) were 30 µg/m^3 and 117 µg/m^3 respectively, with the interquartile range being 20.0 to 41.0 µg/m^3. For $PM_{2.5}$, median and maximum values were 18.0 and 86 µg/m^3 respectively, and for $PM_{2.5}$ sulphate, 2.5 and 15.1 µg/m^3 respectively (n=657-660 days). An increase of 30 µg/m^3 in daily PM_{10} was associated with an odds ratio of 1.22 (95% CI 1.03-1.45) for incidence of cough, 1.53 (95% CI 1.20-1.95) for incidence of lower respiratory symptoms, and 1.22 (95% CI 0.98-1.52) for incidence of upper respiratory symptoms. These increases were similar to those seen in the two Utah valley studies on schoolchildren (Pope et al., 1991; Pope and Dockery 1992). Concentration-response curves for PM_{10} and each of the three endpoints, controlled for temperature, city, and day of the week, were monotonically increasing, and were not significantly different from linear. A 20 µg/m^3 increase in $PM_{2.5}$ was also associated with increased incidence of cough (OR 1.19; 95% CI 1.01-1.42) and lower respiratory symptoms (OR 1.44; 95% CI 1.15-1.82), while a 5 µg/m^3 increase in $PM_{2.5}$ sulphate was associated only with increased incidence of lower respiratory symptoms (OR 1.82; 95% CI 1.28-2.59), and not with increased incidence of cough (OR 1.23; 95% CI 0.95-1.59). Hydrogen ion acidity was not associated with any of the three endpoints. PM_{10} was thus considered to be the best predictor of the particle metrics for increases in adverse respiratory symptoms due to particulate air pollution. In bivariate co-regressions with ozone, SO_2 and NO_2, the association with PM_{10} remained strong and relatively unchanged, while the associations for SO_2 and NO_2 were reduced and no longer significant. Ozone was associated with cough incidence, in the single analysis, and its coefficient remained almost the same, but with a loss of significance in the bivariate analysis, which is interpreted to mean that ozone had some effect on the incidence of cough which was independent of the PM_{10} effect.

12.4.2 Days of Work or School Absences, and Restricted Activity Days

One study attempted to associate PM_{10} with school absenteeism in 6700-6900 elementary schoolchildren from Utah Valley (Ransom and Pope, 1992). A daily time-series was conducted for the school years from 1985 to 1990. The main source of the PM_{10} in this study is a steel mill that was cited for multiple violations of the US EPA particulate standard of 150 µg/m^3 throughout the monitoring period. Mean levels of PM_{10} recorded were 50.9, 50.4 and 40.9 µg/m^3, with maximums of 243, 251 and 234 µg/m^3 for the Lindon, Orem and Provo school districts, respectively. Other pollutants measured included O_3 and SO_2, which were both very low in this area during the school year. Average absenteeism was found to be higher during periods of relatively high PM_{10} pollution than during periods with low PM_{10} pollution. Because of the potential lagged effects of particulates, moving averages of 7, 14, 21, 28 and 35 days were calculated, and the best fit was selected. Absenteeism was approximately 54% and 77% higher on days with a 28-day average above 100 µg/m^3 relative to those days with an average below 50 µg/m^3. However, caution was advised when interpreting the data, owing to the many and varied components that could not be controlled for and which may have influenced absenteeism. Although results from this study show high correlations between absenteeism and elevated levels of PM_{10}, this type of measured effect is more useful as a qualitative indicator of effects rather than in evaluating a dose-response relationship.

The relationship of airborne particulate matter to morbidity, measured as days of work lost, or days of restricted activity, was investigated in a cross-sectional analysis of 68 metropolitan areas in the US (Ostro 1987). A sample of adults, ages 18-65, was taken from metropolitan areas of all sizes and regions of the United States. These cities were part of the Health Interview Survey (HIS) conducted by the National Center for Health Statistics. The HIS is an annual multistage probability survey of individuals in which data on social, economic, demographic and health status are collected. The author used available data for 1976-1981 and a Poisson distribution to model the following health endpoints: days of work loss (WDL), days of restricted activity (RAD) and days of respiratory effects-related restricted activity. Days of restricted activity are defined as days when a respondent was forced to alter his/her normal activity and indicates the most generalized health outcome. The respondents' responses to questionnaires on WDL and RAD in the previous two weeks were correlated to the previous two-week pollutant levels. Data on fine particulates (<2.5 µm) were based on airport visibility data. Particle light scattering, a primary factor in the development of regional hazes, is dominated by particles ranging from 0.1 to 1.0 µm in size (Chapter 7). The PM mass measured by particle light scattering is roughly equivalent to fine particulates, because nearly all of the fine particle mass is from particles less than 1 µm in size. A mean particulate concentration of 24 $\mu g/m^3$ was calculated based on the arithmetic mean of four daytime three-hour readings. The airborne particulates came from a wide variety of urban sources, such as vehicle exhaust and industrial emissions. Regression analysis demonstrated a consistent association between fine particulates and the health endpoints measured. For days of respiratory symptoms-related restricted activity, the estimated coefficient for fine particulates was positive and significant in all six years. A 10 $\mu g/m^3$ increase in estimated fine particles resulted in an estimated 2.8 to 9.0% increase in restricted activity days (RAD) (n=10 900-12 800). For days of work loss, the estimated coefficient of fine particulates was positive and significant in four of the six years and negative in two years; this measure was therefore unstable. The authors noted, however, that the years with the lowest RAD coefficients were also the two that were not significant for working days lost. The health variables in this study may be attributable to a variety of factors. Measurement of fine particulates by airport visibility is a relatively crude means of estimating actual fine particulate levels. Maximum and minimum levels of particulates, which are good relative indicators of air pollution episodes, were not reported (Ostro, 1987).

Another study by Ostro and Rothschild (1989) again used HIS data from the years 1976-1981 for acute respiratory morbidity (respiratory symptoms-related restricted activity days (RRAD)and minor restricted activity days). Two pollutants were analyzed: fine particulates (<2.5 µm, estimated from daily airport visibility data) and ozone. The mean concentrations of fine particulates for each year ranged from 20.7 to 22.3 $\mu g/m^3$. A consistent positive relationship was found between fine particulates and RRAD. The relationship is statistically significant in all six years, with an estimated coefficient ranging from 1.02 to 1.81 in the regressions in which both fine particulates and ozone were included. The association of fine particles with minor restricted activity days was weaker than with RRAD. A 1 $\mu g/m^3$ change in concentration was associated with a 1.58% increase in respiratory symptoms-related activity days, or an excess of 4800 days per 100 000 population.

In an expansion of the data on fine particles, Ostro (1990) repeated the methodology of the previous two surveys, using HIS data from 1979-81, on acute respiratory morbidity, measured as the number of days of RRAD in the two weeks prior to the day of the survey. Health outcomes were compared for TSP (<30 µm), inhalable particulates (IP, <15 µm), fine particles (FP, <2.5 µm) and sulphates (SO_4^{2-}). The total data set included 25 metropolitan areas throughout the United States, concentrated mostly in the south. Data for the 24 h mass of the particles were collected every six days. The mean concentration for fine particulates was 23.5 $\mu g/m^3$, and the correlation between the different measures ranged from 0.81 between FP and IP to 0.35 between TSP and SO_4^{2-}. No maximums and minimums were reported. Results showed that among the particulate measurements used, only the sulphate measurement was positively and significantly associated with respiratory health status, with an 8.3% increase in RRAD per 10 $\mu g/m^3$ increase in SO_4^{2-}. Inclusion of a two week lag period (in addition to a two week mean) increased the RRAD by 17.4% for SO_4^{2-}, 12.9% for FP, 9.0% for IP, and 6.4% for TSP (all significant at p=0.01) (Ostro, 1990). Tnis study provides good support for the effect of fine particles compared to IP or TSP, the relative strength of association being SO_4^{2-} >FP >PM_{10} >TSP. The results were not robust for winter, when ozone levels were low, suggesting that at least part of the effect could have been due to an independent or combined effect of ozone, an unmeasured confounder.

12.5 LONG-TERM AND CHRONIC EFFECTS

The effect of longer term exposure (i.e., greater than daily or weekly variations) or chronic exposure has been examined by means of cross-sectional studies that compare populations in different geographic regions with different pollutant mixes, and cohort studies on a defined group of individuals followed through time. Most of these studies, including some with the cohort design, have had to rely on grouped community exposures rather than individual exposures, although they may have data on other individual risk factors such as smoking or occupational history. Studies have been grouped according to endpoint, on mortality, long-term effects on the respiratory system, and cancer.

12.5.1 Cross-Sectional and Prospective Studies on Mortality

A number of cross-sectional studies, most carried out prior to 1990, have been reviewed by Lipfert and Wyzga (1995b), Pope et al. (1995a-c), and the US EPA (1996). The general conclusions were that although mortality rates were associated with air pollution, most strongly with fine particles or sulphate, the study limitations were sufficiently great (limited air quality data, subject to confounding by smoking, lifestyle factors, and choice of model), that the studies were in fact of limited value in the elucidation of the mortality – air pollution/particulate matter association. Only one of these population-based cross-sectional studies (Ozkaynak and Thurston 1987) is considered here, because it investigated several fine particle associations which are potentially of greater concern than larger particle size fractions. Two large cohort studies have also recently been completed. Since these were able to include individual risk factors known to affect mortality and they followed individuals through time, they are considered to provide better and more reliable evidence than the cross-sectional studies on the pollution – mortality association. One study (Pope et al., 1995a) was based on a large sample of participants in the American Cancer Society's cohort study covering 150 US communities while the other (Dockery et al., 1993) was based on a more limited sample from six US cities. Both studies considered at least two measures of fine particles, as well as larger particle sizes including PM_{10} and TSP.

In the cross-sectional mortality study by Özkaynak and Thurston (1987), fine particles (FP or $PM_{2.5}$) and inhalable particles (IP or PM_{15}) were estimated for 60 of the 98 US Standard Metropolitan Statistical Areas (SMSAs) which did not have measured values. The estimating techniques gave ± 30% error for IP and ± 13% error for FP. TSP and SO_4^{2-} measurements were also collected, but no gaseous pollutants were considered. Mortality rates from the year 1980 were extracted for 98 areas from the 117 SMSA data set, and a regression analysis was conducted on the association between the annual averages of various particle measures and total mortality, including terms for median age, the proportion of population over age 65, proportion of non-whites, population density, proportion with a college education, and proportion below the poverty line. No correction for temperature was employed.

Of the particle measurements examined in the study, SO_4^{2-} showed the strongest and most consistent association with increased mortality, followed by fine particulate matter. Estimates for various regression models ranged from 6.1 to 7.5 deaths per 10^5 per $\mu g/m^3$ for SO_4^{2-}, and 2.1-3.7 deaths per 10^5 per $\mu g/m^3$ for FP (both measured and estimated). This was equivalent to an 8.2% increase in deaths per year per 10 $\mu g/m^3$ for SO_4^{2-} (95% CI 5.0-11.4%), and a 4.3% increase for FP (95% CI 0.4-8.2%) (single regression). The estimated mean concentrations of sulphates and fine particulate matter were 11 and 23 $\mu g/m^3$ respectively, and estimated total mortality effects of air pollution ranged from approximately 4 to 9% for SO_4^{2-} and 3 to 8% for $PM_{2.5}$. The association between mortality and inhalable particles (IP or PM_{15} and TSP) was of lower magnitude, at 0.8% (95% CI 0.14-1.5%) and was not a significant predictor of mortality. In a model with both estimated and measured values for FP and IP, the regression coefficients were strengthened for the measured versions of both FP and IP, but IP remained below the level of statistical significance. The measurement error noted above could have been responsible at least in part for the non-significance of IP. Overall, the pollution coefficients had the ranking $SO_4^{2-} > FP > PM_{10} > TSP$. Simultaneous regression including all four particulate metrics indicated that only SO_4^{2-} was significantly associated with mortality, but the high correlations between SO_4^{2-} and FP casts some doubt on the lack of positive results with respect to FP. Correlations between SO_4^{2-} and TSP or IP were low, -0.06 and 0.14 respectively. The analysis was sensitive to the results from the Ohio Valley, where sulphates are high; removal of the Ohio Valley results lowered the SO_4^{2-} regression coefficient by 40-50%.

The nature of the study did not allow for consideration of confounding factors such as smoking. However, sensitivity analyses showed that the overall statistical trend and conclusions were relatively robust to model adjustments. An interesting analysis of particle sources was carried out for 36 of the SMSA areas (Ozkaynak and Thurston 1987). Small particles from the iron/steel industry and coal combustion appeared to be relatively greater contributors to mortality (regression coeff. 10.0 and 5.7 deaths/10^5/ $\mu g/m^3$ respectively) than the larger soil-derived particles (regr. coeff. 0.7, not significant).

Mortality risks associated with air pollution were studied in a prospective cohort study beginning in 1974 (Dockery et al., 1993). Data were obtained from a 14- to 16-year mortality follow-up of a random sample of 8111 adults (1430 deaths) from the Harvard Six Cities Study (Watertown, Massachusetts; St. Louis, Missouri; Portage, Wisconsin; Kingston-Harriman, Tennessee; Steubenville, Ohio; Topeka, Kansas). Measurements were taken for five classes of particles: TSP, fine particles (<2.5 μm), inhalable particles (<15 μm before 1984 and <10 μm starting in 1984), SO_4^{2-}, and H^+. Other pollutants included were NO_2, SO_2 and O_3. At the beginning of the study, spirometric tests were administered, and information was collected by questionnaire on age, sex, weight, height, education level, complete smoking history, possible occupational exposures, and medical history. After adjustment for age (5 y categories), body-mass index, current and former smoking, number of pack-years of smoking, and an indicator for less than a high-school education (as a surrogate for socioeconomic status), significant and robust associations were observed between air pollution and mortality, as assessed by comparison of the range of pollutants in the most polluted versus the least polluted city. Based on the authors' graph (figure 2 of Dockery et al., 1993), the probability of survival over a 14-year period was reduced from 88% in the least polluted city to 79% in the most polluted city, and represented an average life-shortening of about four years (the mean for all six cities thus being two years). This strongly suggests that the effect on mortality is not just a phenomenon of "harvesting" or hastening the deaths of seriously ill people by a few days or weeks.

Mortality risk was most strongly associated with mean concentrations of inhalable particles ($PM_{10/15}$) (mean concentration range 18.2-46.5 μg/m^3), fine particles ($PM_{2.5}$) (mean concentration range 11.0-29.6 μg/m^3), and sulphate particles (4.8-12.8 μg/m^3) (Dockery et al., 1993). The adjusted rate ratios (RR) for these ranges were virtually the same at 1.27 (95% CI 1.08-1.48) for $PM_{10/15}$, 1.26 (95% CI 1.08-1.47) for $PM_{2.5}$, and 1.26 (95% CI 1.08-1.47) for SO_4^{2-}. This was equivalent to 10%, 14% and 32% increases in the chances of dying for each 10 μg/m^3 increase in PM_{15}, $PM_{2.5}$, and SO_4^{2-} respectively. Ratios were smaller for TSP, SO_2, and NO_2. These associations persisted after stratification by the individual city. In an additional analysis of these data (US EPA 1996), the RR for TSP was 1.22 and was marginally significant (lower confidence limit (LCL) =0.99), but when TSP data from 1970, prior to the beginning of the study, was regressed, the RR increased to 1.25 and was significant (95% CI 1.03-1.50). This, taken with the information that TSP pollutant levels had decreased over the years, suggests the importance of previous exposure. The EPA re-analysis also investigated the importance of coarse particles, as PM_{15}-$PM_{2.5}$, and the sulphate portion of the fine fraction, as $PM_{2.5}$-SO_4^{2-}. The coarse fraction was not significant (RR 1.19; 95% CI 0.91-1.55), but the non-sulphate fraction of $PM_{2.5}$ retained significance (RR=1.24; 95% CI 1.16-1.32), and the regression co-efficient doubled from the $PM_{2.5}$ value and dropped about 14% from the SO_4^{2-} value. The lack of significance of acidity (H^+) is to be noted, since Kingston-Harriman, TN was selected for the study on the basis of its high acidity levels. However, only one year of measurements were available. Ozone was not significant, but 24 h measurements were used instead of peak daytime levels, and ozone levels did not differ much between cities; either or both of these factors could have been responsible for the lack of association with ozone. Although a supplementary analysis of these data by the US EPA (1996), showed that SO_2 and NO_2 were associated with premature mortality at least as strongly as TSP in single pollutant regressions (rate ratios 1.20 with 95% CI 1.01-1.43 for SO_2 and 1.22 with 95% CI 1.00-1.49 for NO_2, these associations were of smaller magnitude and were less robust than the associations with PM_{15}, $PM_{2.5}$, SO_4^{2-}, and the non-sulphate fraction of $PM_{2.5}$. Thus SO_2 and NO_2 were unlikely to act as confounders in the particulate matter associations despite the lack of testing in bivariate regressions.

A strong linear concentration-response was shown in the US EPA reanalysis (1996) of the Dockery et al. (1993) data for sulphate and non-sulphate fine particles and for fine particles combined. The linear association was a little weaker for inhalable particles (PM_{15}) and the coarse particle fraction of inhalable particles (PM_{15}-$PM_{2.5}$). TSP or total particles and the non-inhalable fraction (TSP-PM_{15}) showed only a weak linear trend. However, it is noted that this is based on only six observations (cities). Exposure misclassification could have accounted for the reduced significance of TSP and coarse particles, since they are less homogeneous over the six areas, which were represented by only one monitoring location each. Additionally, these do not penetrate as readily indoors (where most people spend most of their time) as the finer particles and sulphates.

The mortality effect of individual risk factors including current or former smoking, 10 or 25 pack-years of smoking, less than a high school education in men, body-mass index in women, and living in the most polluted city (Steubenville), was analyzed in a combined Cox proportional hazards model. The largest RR was observed for current smokers (RR= 1.59; 95% CI 1.3-1.9). The estimated risk ratio between the most and least polluted city using $PM_{2.5}$ as an indicator was 1.26, with a 95% confidence interval of 1.08-1.47. The rate ratios for the effect of $PM_{2.5}$ on mortality among former and current smokers, and for men with occupational exposure, were slightly increased to 1.32-1.35, indicating that these factors may have had some influence on the increase in risk. By category of cause of death, risks in the most polluted versus the least polluted city were increased for cardiopulmonary disease (RR=1.37) and lung cancer (RR=1.36), but the latter was not statistically significant. Exclusion of persons with pre-existing hypertension or diabetes did not lower the RR. Overall, this study found a strong and consistent relationship between increased mortality and particulate matter. This relationship was robust to various model specifications and to the inclusion of several possible confounders (Dockery et al., 1993). Because of the lack of long-term monitoring for most of the pollutants prior to the start of the study, the potential effect of chronic non-concurrent exposure could not be investigated.

A second large prospective cohort mortality study has been carried out on a much larger (non-random and not entirely representative) sample of the US population (Pope et al. 1995a). This study used data on individual risk factors from a cohort of 552 000 adults aged 30 or older (the ACS or American Cancer Society Cohort) who resided in 1980 in 151 US metropolitan areas. Deaths (38 000+) were recorded for 7.3 years from September 1982 to December 1989. Death certificates were obtained for 96% of all deaths and coded to ICD-9 by a specialist. A subcohort (295 000 subjects; 20 765 deaths) was also selected from 50 cities which had available fine particle data in addition to the SO_4^{2-} data for the main cohort. Pollutant concentrations for the year 1980 were used as the index of exposure for sulphate (mean 11.0 $\mu g/m^3$; range 3.6-23.5 $\mu g/m^3$), and the median fine particle concentration for 1979-1983 from a previous cross-sectional study (Lipfert et al., 1988) was used for the subcohort (median average $PM_{2.5}$ = 18.2 $\mu g/m^3$; range 9.0-33.5 $\mu g/m^3$). Relative risk ratios, stratified by age (5 y intervals), sex, race, and gender, and adjusted for smoking status, passive smoke exposure, duration of exposure, body-mass index, alcoholic drinks, educational attainment, and occupational exposure, were calculated based on the Cox proportional hazards model using survival time as the variable. All information had been collected by interview prior to the starting date.

For individuals residing in the city with the highest mean pollutant levels compared to those with the lowest levels, the risk ratio of dying over the 7+ year period was 1.15 (95% CI 1.09-1.22) for an increased sulphate level of 19.9 $\mu g/m^3$ (its range over all cities), and 1.17 (95% CI 1.09-1.26) for increased $PM_{2.5}$ were of 24.5 $\mu g/m^3$ (also its range). Per 10 $\mu g/m^3$ increase, this was equivalent to a 7.5% increase in the chance of dying in the high sulphate area or to a 6.9% increase in the high $PM_{2.5}$ area. The risk ratio was increased for both sulphates (RR =1.26; CI 1.16-1.37) and $PM_{2.5}$ (RR=1.31; 95% CI 1.17-1.46) for cardiopulmonary deaths. Increased lung cancer deaths (RR=1.36; 95% CI 1.11-1.66) were associated with sulphates but not with $PM_{2.5}$ (RR=1.03; n.s.- LCL 0.80). These RRs were internally consistent with the null results for all other causes of death (RR= 1.01 for SO_4^{2-} and 1.07 for $PM_{2.5}$, neither significant), and with the greatly increased RR observed in all four categories for current smokers who smoked one pack of cigarettes per day for 25 years or more (e.g., the lung cancer RR associated with smoking was 9.73 for a person who smoked one pack per day for 25 years. A broader range of individual risk factors, especially those having to do with smoking status, was included in this study than in the Dockery et al. (1993) cohort study. The associations between mortality and particulate air pollution were not sensitive to the inclusion of body-mass index (BMI), alcohol consumption, education or occupational exposures. Temperature effects could not explain these increases in risk ratio, since temperature highs, means or lows were not correlated with sulphates or fine particles ($r<0.1$) and analyses including variables for "hot" (mean >60°F) and "cold" cities (<50°F) had little impact on the results. No other air pollutants were considered in the analysis, but the wide geographic range of cities covered in the analysis would include a broad range of air pollutant mixes, making less likely the possibility that the effects were due entirely to confounding.

A population-based cross-sectional ecological analysis was also carried out for the same cities or metropolitan areas, for comparison with the cohort study

(Pope et al., 1995a). Age-, sex-, and race-adjusted regression coefficients for total mortality were 10.5 ± 1.3 deaths/year/100 000 people per µg/m^3 SO_4, and 8.0 ± 1.4 deaths/year/100 000 people per µg/m^3 $PM_{2.5}$ ($p<0.001$). No other lifestyle confounders (e.g., smoking status) were included. Using the mean age-sex-race adjusted mortality rate as the baseline risk, the estimated risk ratios for the most polluted city versus the least polluted city were 1.25 using sulphate and 1.24 using $PM_{2.5}$. These were somewhat higher than the RR found in the prospective cohort study, of 1.15 for SO_4^{2-} and 1.17 for $PM_{2.5}$. Thus the inclusion of likely demographic and lifestyle confounders in the cohort study reduced the RR due to pollution but did not eliminate it. The coefficents were also almost twice as high for SO_4^{2-} and two to four times higher for fine particle coefficients as the coefficents found in the cross-sectional ecological analysis by Ozkaynak and Thurston (1987). The risk ratios in the main cohort study were consistent with, but somewhat lower than those seen in the six-city study. This was probably due to the much larger geographic and population coverage of 151 cities versus six cities, as well as to inclusion of a broader range of potential confounders in the regressions. These risk ratios (Pope et al., 1995a) are therefore considered to be a more accurate expression of the relationships of mortality to sulphate and fine particles, as combustion-source air pollutants. It is noted that the observed risk ratios may also be a consequence of higher pollutant concentration in earlier years, prior to the 1980 determination of exposure levels, a hypothesis supported by the higher risk ratios associated with 1973 TSP levels.

12.5.2 Long-Term Effects on Respiratory Health and Pulmonary Function

This section considers cohort and cross-sectional analytical studies that have considered a range of particle metrics in examining the relationship of seasonal or annual variations in air pollution to worsening of respiratory symptoms and/or lung function. Included are two recent and two older cross-sectional studies on children, as well as a British study on a birth cohort of young adults and a prospective cohort study on older adults in California in which individual exposure estimates could be made.

In a cross-sectional study carried out in the 1978-79 school year, pulmonary function and respiratory symptoms in children aged 8 to 10 years (n=300 to 400 for each community and season) from five Montana communities were examined for possible associations with seasonal variations in TSP, and in fine and coarse particles (Johnson et al., 1990). Air pollution, which was highest in winter, arose from industrial sources (mining, smelting, pulp and paper mill, sawmill, coal-fired power plant) and both industrial and residential wood-burning. The three-month seasonal averages for TSP in the five cities ranged from 27 to 128 µg/m^3, for coarse particles ($PM_{15-2.5}$) from 11 to 56 µg/m^3, and for fine particles ($PM_{2.5}$) from 8 to 46 µg/m^3 . Differences between cities were more striking with TSP than with fine or coarse particles. TSP was measured daily, while fine and coarse particles were measured only every third day and the remaining two days estimated. Correlation coefficients for TSP – coarse particles, TSP – fine particles, and TSP – fine plus coarse particles (PM_{15}) were 0.82, 0.55, and 0.32 respectively (the latter being unexpectedly low). Pulmonary function tests (FEV_1, FVC, FEF_{25-75}) were carried out in each of the cities three times over the study period, once each season, and the results were analyzed by season. Great Falls children showed the highest pulmonary function values, and these were used as controls for the other four cities. Results for FEV_1/FVC and for FEF_{25-75} were the lowest in Anaconda and Butte, followed by Missoula. FEV_1 and FVC were consistently lowest in Missoula. Researchers observed the greatest negative correlations between TSP and PM_{15} and pulmonary variables in simple linear regression analysis, but the detailed results were not presented. The slope for FVC for all females was -0.39 ml/µg/m^3 and for males it was -0.65 ml/µg/m^3, roughly similar to the results of other researchers, but the authors noted that the significance level was low for girls. The effects observed also appeared to be transient: children's pulmonary function rose significantly after two months of clean spring air. (Johnson et al., 1990). The analysis and reporting of the pollution-response data was vague and inadequate in this study. Although this study does not support a greater role for fine particles than for coarse particles, this could be a function of the unsophisticated analysis as well as inaccurate measurement and estimation.

Dockery et al. (1989) conducted a cross-sectional study of air pollution and health using a health questionnaire and spirometric measurements. The subjects were 5422 children aged 10-12 years in six cities (Watertown, Massachusetts; St. Louis, Missouri; Portage, Wisconsin; Kingston-Harriman, Tennessee; Steubenville, Ohio; and Topeka, Kansas) in the eastern United States. The sources of pollution again included vehicle exhaust and industrial emissions. Pollutants measured were $PM_{2.5}$, PM_{15}, TSP, SO_4^{2-}, SO_2, NO_2 and O_3. Mean levels reported for $PM_{2.5}$ ranged from 11.8 to 36.7 µg/m^3. Health data collected during the 1980-1981 school year included the following five respiratory illness and symptom responses: bronchitis, cough, chest illness, wheeze and asthma. Pulmonary measurements recorded were FVC, FEV_1, $FEV_{0.75}$ and MMEF.

The only significant associations found between measures of particulate matter and measures of illness and symptoms were with bronchitis, and possibly chronic cough. The odds of contracting bronchitis over the range of PM_{15} observed (38.7 µg/m^3) were increased by a factor of 2.5 (with a 95% confidence interval of 1.1-6.1 µg/m^3). Although the odds ratios were in the same range for the other particle metrics, they were not significant; for example, the OR for $PM_{2.5}$ (range 24 µg/m^3) was increased by a factor of 2.1 with a 95% confidence interval of 0.8-5.9. These non-significant results appeared to be due to the low power of the study to detect effects of this size, given that there were only six units of observation (i.e., the pollution level in each of six cities). It was also observed that among the 571 children with asthma or persistent wheeze, the prevalence of bronchitis, and to a more limited extent, chest illnesses and chronic cough, was much higher than in children without these symptoms. When the prevalence of bronchitis was plotted against the city-specific mean concentrations of PM_{15}, it became clear that children with asthma were reporting most of the excess cases of bronchitis in the more polluted communities, since the slope of the regression curve was close to zero for the non-asthmatics. This association was found for all four particle measurements, but was strongest for PM_{15}. There was no evidence of adverse effects on pulmonary function associated with any measure of particulate air pollution as experienced over a period of one year, although this could have been due to the low power of this study to detect such effects (Dockery et al., 1989).

The same research group has recently completed a much larger cross sectional analytical study to investigate the relationship of fine particles, ozone, and acidity with both lung function and respiratory symptoms in children of 24 communities in the US and Canada (Dockery et al., 1996a; Raizenne et al., 1996). Communities were selected on the basis of provision of a wide variety of demographic and atmospheric conditions, and were largely suburban or rural, without significant industrial sources of pollution. Eleven communities (all US) were in the "sulphate belt", six were in the "transport region" for sulphates and photochemical smog (four in southern Ontario), three were west coast (California) and four were "background" (including Yorkton, Sask. and Penticton, B.C.). For the respiratory symptoms study, 13 369 nine- and ten-year old (white) school children in 24 communities participated, with the median being 581 per community (range 329-676) (Dockery et al., 1996). One-hour maximum ozone (mean 46.5 ppb), SO_2 (mean 4.8 ppb), ammonia (mean 1.3 ppb), and gaseous acids (nitric and nitrous acid, mean 59 nmol/m^3) were monitored in addition to PM_{10} (mean 23.8 µg/m^3), $PM_{2.1}$ (mean 14.5 µg/m^3), SO_4^{2-} (mean 4.7 µg/m^3) and H^+ (mean 31.1 nmol/m^3). Monitoring began for this study in the spring of the preceeding school year and continued for at least one year (study was carried out in the different communities over three years, 1988-1990). This one year of monitoring was shown to be representative of a lifetime of exposure for most pollutants, and for a majority of the children, based on at least four years of monitoring results, available for PM_{10}, $PM_{2.1}$, and SO_4^{2-}, but only one year for particle strong acidity (PSA or H^+); a detailed exposure assessment and discussion of the monitoring results was reported by Spengler et al. (1996). A standard respiratory symptom questionnaire on symptoms in the past 12 months was filled out by the parent(s) in the fall (average response rate 91%) and lung function tests were administered at the school later in the year.

After adjustments on a city-specific basis for sex, child's history of diagnosed allergies, parental history of diagnosed asthma, parental (either parent's) post-high school education, and current smoking in the home, the odds ratio for developing bronchitis in the past year were elevated for communities with high acidity and high sulphate levels, the OR for a PSA range of 52 nmol/m^3 from the lowest to the highest community being 1.66 (95% CI 1.11-2.48), and the OR for a sulphate range of 7 µg/m^3 being 1.65 (95% CI 1.12-2.42). Respirable particulates ($PM_{2.5}$; range 14.9 µg/m^3) and inhalable particulates (PM_{10}; range 17.3 µg/m^3) were associated with increased bronchitis (OR=1.50) but the association was not significant. Gaseous acids (nitrous and nitric acid) were associated with a higher risk of asthma (OR =2.00; 95% CI 1.14-3.53), as well as marginally elevated ORs for other asthmatic symptoms and chronic phlegm. However, this association was driven by one community with unusually high levels, and the association was no longer significant if this community was removed from the analysis. SO_2 (12.7 ppb range) was associated with increased odds for chronic phlegm (OR= 1.55; 95% CI 1.01-2.37) and marginally associated with bronchitis and other bronchitic symptoms. The maximum 1 h ozone average was not associated with increased odds for any symptoms.

Stratification of the results was carried out by sex, history of a severe chest illness before the age of 2, use of a humidifier in the home, lung function in the lowest quartile of FVC, current exposure to environmental tobacco smoke, prenatal exposure to maternal smoking, year of study, country, and eastern versus western cities. Reporting of chest illness before age 2 years, asthma symptoms, and use of home humidifiers were associated with slight increases (not statistically significant) in the OR for bronchitis associated with acidity, while parental asthma, maternal smoking during pregnancy, and environmental tobacco smoke in the home lowered the OR and reduced its significance. There was no evidence that the prevalence of asthma or asthmatic symptoms in children was associated with air pollution. The strong correlations between particle strong acidity and other particle measures make it difficult to ascribe the effects with certainty to any one of these (PSA-SO_4^{2-} r=0.90; PSA-$PM_{2.1}$ r=0.82). The correlation with ozone was relatively low by study design (r= 0.37). Thus the negative results for ozone can not be ascribed to allocation to particulate measures. The finding that bronchitis was most strongly affected by air pollution is consistent with the previous findings from the six-city study (Dockery et al., 1989), but the agent most closely associated with this was acidity in this study (Dockery et al., 1996a) instead of PM_{10} as in the smaller six-city study.

Lung function testing, including forced vital capacity (FVC), forced expiratory volume (FEV) in 1 and 0.75 sec., forced expiratory flow rate between 25 and 75% of FVC (FEF_{25-75}), and peak expiratory flow rate (PEFR), was carried out in 22 of the 24 communities with a total of 10 251 children providing at least two acceptable tests (Raizenne et al., 1996). After adjustment for height, weight, age and sex, 2 to 3.5% decrements in FVC, FEV_1 and $FEV_{.75}$ were associated with all the particle measurements, with the most marked effects for particle strong acidity (PSA), followed by $PM_{2.1}$, SO_4^{2-}, and PM_{10} in that order. FVC was most strongly affected, and PSA was associated with a 3.5% decrement (95% CI -4.9 to -2.0%) for a range increase of 52 nmol/m^3. Sulphate, $PM_{2.1}$, and PM_{10} were associated with decrements in FVC of 3.1%, 3.2% and 2.4% respectively, for range increases of 6.8 μg/m^3 (SO_4^{2-}), 14.9 μg/m^3 ($PM_{2.1}$), and 23.8 μg/m^3 (PM_{10}). When converted to a standard range increase of 10 μg/m^3 , FVC was reduced 4.5% (SO_4^{2-}), 2.2% ($PM_{2.1}$), and 1.4% (PM_{10}) respectively. The association of lung function decrements with particle strong acidity was consistent throughout the results. Only particle strong acidity was associated with decrements in FEF_{25-75} and PEFR, the associations with the other particle metrics being non-significant. However, the strong correlations between particle strong acidity and other particle metrics (e.g., PSA/SO_4^{2-} r=0.83-0.95 in east and central North America) limits our ability to distinguish which of these, if any, was independently responsible for the observed effect.

Ozone (all three measurements, 24 h, daytime, and 1 h max.) was also associated with decrements in FVC and FEV_1 (Raizenne et al., 1996). A range of 39.4 ppb daytime ozone resulted in a 3.74% decrease in FVC (95% CI -6.45 to -0.94%). Bivariate regression with PSA reduced this association to -2.2%, but significance was retained. The PSA association remained unchanged in the co-regression. This indicates that ozone has some independent association with decrements in lung function, additional to the association with PSA, but it was noted that the PSA-ozone correlation was moderate to high (r=0.45-0.61 for 1 h max., average of all communities except four Prairie communities).

No potentially sensitive subgroups in the population were identified when regressions were carried out with subgroups of children who were asthmatic, wheezy, who had had severe chest illnesses before age two, whose parent(s) had asthma (although the association here was slightly stronger), or who lived in homes with smokers. This was based on lack of appreciable differences in decrements in FVC between any subgroups and the overall mean, although all were significantly different from null. Air pollution was associated with low lung function in some children, whose measured FVC was <85% of predicted for their age, sex, and size. PSA across the 52 nmol/m^3 range of exposures was associated with a 2.5 times increase in the proportion of children whose FVC was <85% (95% CI 1.8-3.6%). This proportion was not altered by bivariate regression with ozone, but the odds of having a FVC <85% associated with the range of 38.4 ppb daytime ozone were reduced from

2.9% (95% CI 1.4-5.8) with ozone alone, to 2.0% (95% CI 1.6-2.5%) for ozone with PSA included in the regression. This represents a significant decrement in lung capacity and lung growth, development, and function in children, associated with chronic exposure to the air pollutants ozone and PSA (and probably also with several of the other particle metrics) (Raizenne et al., 1996). The data analysis carried out for this study tended to emphasize the results for particle strong acidity, without presenting the same depth of analysis for $PM_{2.5}$ or SO_4^{2-}, each of which had almost as strong an association with decreases in FVC, FEV_1 and $FEV_{0.75}$ as particle strong acidity.

A possible explanation for the lung function decrements is that air quality on the day of testing or on the several previous days was poor, and was the cause of the observed decrements. Since so many tests were conducted in a broad range of geographic locations on many different days, this is not a likely explanation for the majority of observations. However, the year of testing could also have had unusually high air pollution, which would have affected the results for some communities compared to others, since about one-third of the testing was carried out in each of the three different years in the various communities. The chronic nature of the effects was explored in several ways. The annual averages for the communities were closely comparable to regional results for 4-13 years of monitoring data for all particle metrics (except H^+, for which only one year was available). This indicates that they did represent chronic exposures (Spengler et al., 1996). Within the study itself, stratification by residence in the community since age 6 and residency since before age 2 showed that lifelong residency provided stronger links with FVC decrements (4.2%; 95% CI 2.5-5.8%) than the shorter residency of children who had moved into the community more recently, within the past three to four years (FVC -2.4%; 95% CI 1.0-4.0%).

A British cross-sectional study investigated the effect of current annual levels of air pollution, as BS and SO_2 on the prevalence of respiratory symptoms in a birth cohort of 11 552 23-year old young adults who had been born in March 1958 and interviewed in 1981 (Scarlett et al., 1995). Exposure status was by population only, on a county-wide basis (n=53). Counties were further grouped, roughly into quintiles for both measurements. Mean BS was 17.9 μg/m^3 (range 2.0-55.1 μg/m^3), and the medians of the quintiles were 11.0, 16.4, 20.5, 22.3, and 30.2 μg/m^3. Mean SO_2 was 42.6 μg/m^3 (range 7.0-87.7 μg/m^3), and medians of the quintiles ranges from 43.0 to 50.5 μg/m^3. After adjustment for sex, social class, and smoking status, the odds ratio for prevalence of chronic phlegm was increased in all four groups (ORs = 1.36, 1.34, 1.48, 1.20) relative to the lowest group for which the median was 11.0 μg/m^3 ($p<0.01$), and the test for trend was significant. The relationship was, however, somewhat non-linear, with the OR (1.20) for the highest group lower than expected, indicating some offsetting factor in this group, and/or the possibility of a flattening of the concentration-response curve. BS had no effect on the prevalence of wheeze or cough symptoms, nor on asthma prevalence either at age 16 or at age 23. There was no evidence that SO_2 independently influenced the prevalence of any of the symptoms, but BS and SO_2 were highly correlated (r=0.61), which makes separation of their effects difficult. Smoking exerted a much greater effect on cough and phlegm prevalence and on asthma at age 16 than the air pollution indices. This study indicates that chronic or long-term exposure to particulate air pollution, as measured over the preceding year, increases the prevalence of some respiratory symptoms (not including asthma) in young adults, but that the air pollution effect was considerably less than the effect of smoking. The study could neither confirm nor refute the hypothesis that the observed effects were a residual of exposure in childhood, since few ambient measurements were available for that early period.

A longitudinal study on respiratory symptoms and lung function changes over a 12-year period was carried out with a random sample of 3948 adults aged 25 to 74 years in 1974, from the six US cities cited in other analyses (Sherman et al., 1992). At the initial and followup visits after three, six, and 12 years, subjects completed a respiratory symptom questionnaire and lung function tests (forced expiratory volume in one second – FEV_1). Sex-specific linear regression models were developed to investigate the relationship between symptoms and lung function. After adjustment for height, age and smoking status, those who reported chronic symptoms of cough and/or phlegm on the inital visit showed both a slight reduction in initial lung function and an accelerated decline in lung function. The difference between observed and predicted slope for subjects with any critical respiratory symptoms was -4.0 ml/y for males and -2.5 ml/y for females ($p>0.05$), and for cough, the decline was 5.6 ml/y for males and 4.4 ml/y for females ($p<0.001$). The effect was likely to be highly underestimated because of the high attrition in the original cohort (52% of cohort not included) and the pronounced "survivor" effect in those who could be included. While this study does not deal directly with particulate matter-lung function associations, it sheds some light on the relationship between the prevalence of chronic symptoms and acceleration in lung function decline over time.

The effect of air pollution over a 10- to 20-year period on non-smoking adults has been examined prospectively in a cohort of non-smoking adults in California (Abbey et al., 1995a). A subcohort of 1868 individuals from the AHSMOG cohort (Adventist Health and Smog Study) who had resided since 1966 within a few miles of nine California airports were selected, and their individual cumulative exposure to $PM_{2.5}$ was calculated, based on site and season-specific estimates of $PM_{2.5}$ from airport visibility data (r=0.82), and adjusted individually by taking into account their duration of exposure, and monthly residence histories. TSP, PM_{10} estimated from TSP, SO_4^{2-}, O_3, SO_2, and NO_2 data were also estimated for each individual. A standardized respiratory questionnaire was given in 1977, and again in 1987, to all participants who had resided in their area for the past 10 years since 1966. The questionnaire also ascertained household and lifestyle characteristics (gender, age, educational status as a surrogate for socioeconomic status, occupational exposure, any previous smoking history (Adventists are non-smokers), passive smoking exposure, and history of childhood colds and/or childhood airway obstructive disease (AOD). The development between 1977 and 1987 of new cases of respiratory symptoms of AOD, chronic bronchitis, and asthma was followed in those without possible symptoms in 1977, and correlated with their previous chronic exposure in the 10 years preceding the start of the study, as well as their "current" 10 year average exposure from 1977 to 1987. The relative risk (RR) of developing new cases of chronic bronchitis was elevated at 1.81 (95% CI 0.98-3.25; p=0.058) for an increment of 45 $\mu g/m^3$ in average $PM_{2.5}$ in the years 1966-March 1977. This is equivalent to an 18% increase per 10 $\mu g/m^3$. Slight enlargement of the cohort to 1940 subjects who had resided only 80% of time instead of 100% in the same location in the 1966-77 period resulted in statistical significance being reached. An increased relative risk was also associated with an exceedance cutoff of 20 $\mu g/m^3$, a RR of 1.41 being associated with 4000 h/y (167 d/y) at or above 20 $\mu g/m^3$. A dose-response relationship was observed for new cases of chronic bronchitis, with a 53% increase in new cases for a $PM_{2.5}$ cutoff of <30 $\mu g/m^3$ versus <20 $\mu g/m^3$, and 64% for a 40 $\mu g/m^3$ cutoff versus a 20 $\mu g/m^3$ cutoff (Abbey et al., 1995a). No associations were observed between past $PM_{2.5}$ concentrations and incidence of new cases of asthma or airway obstructive disease (including emphysema, asthma and bronchitis).

The association of new cases of chronic bronchitis with past exposure was stronger than the association with the average 10-year current exposure (1977-87); the RR for the latter was not given but the t=statistic was reduced from 1.90 to 1.68. Current exposure (1977-87) to sulphate was associated with development of new cases of AOD, but not bronchitis or asthma (no measurements were available for past exposure to sulphate, 1966-1977). The development of airway obstructive disease (AOD) and/or asthma was not significantly associated with current exposure to $PM_{2.5}$, but AOD development was significantly associated with past exposure (1966-77) to TSP and PM_{10} (t=2.81 for TSP and 2.09 for PM_{10}).

The presence of possible symptoms in 1977 was highly predictive of increased RR for new cases between 1977 and 1987 of all three conditions, particularly for asthma, in which the RR was almost 26 times higher than unity. A history of childhood colds and childhood AOD also was associated with significant increases in risk for AOD and chronic bronchitis, but not for asthma.

The possibility that $PM_{2.5}$ was acting as a surrogate for one or more other air pollutants was investigated by substituting TSP, PM_{10}, SO_4^{2-}, O_3, and visibility (an indirect measure of fine particles which was used to estimate $PM_{2.5}$) into the regression analyses in place of $PM_{2.5}$ (Abbey et al., 1995a). While "current" 1977-87 SO_4 showed an association with new cases of AOD (t=2.09), and with exacerbations in symptoms of AOD and bronchitis, it was not associated with new cases of bronchitis, thus indicating that it was not a surrogate in the $PM_{2.5}$-bronchitis association ($PM_{2.5}/SO_4^{2-}$ correlation coefficient r=0.35, low-moderate). Past exposure (1973-1977) to PM_{10} and TSP gave stronger associations than $PM_{2.5}$ with development of new cases of AOD (t=2.09 and 2.81 vs. 1.37 for $PM_{2.5}$) and chronic bronchitis (t=2.08, 2.24 vs. 1.90 for $PM_{2.5}$). High correlations between these three metrics (r=0.91-0.92) made it impossible to distinguish between their effects. Ozone exposure (1977-87) was not associated with development of new cases of AOD, bronchitis or asthma, although it was strongly associated, along with all the particle metrics, in exacerbation of asthma symptoms. Its high correlation coefficients with all the PM metrics (r=0.67-0.77) except SO_4^{2-} (r=0.38) makes it difficult to distinguish an independent effect for ozone.

Significant increases in the severity of respiratory symptoms over the 10-year 1977-87 period were also recorded for all three endpoints, in association with past 1966-77 PM_{10} exposure, except for asthma for which present 10-year exposure was more highly associated than past exposure. A number of other factors including the presence of possible symptoms in 1977, AOD in childhood, larger number of childhood colds than average, and past smoking history, were more important than air pollution in exacerbation of symptoms, however. An extensive discussion of the study limitations and sensitivity analyses to address some of these were presented. The authors' conclusions were that previous exposure to $PM_{2.5}$ >20 μg/m^3 was associated with development of new cases of chronic bronchitis, but that the associations with increasing severity of disease could have been due to surrogate relationships with TSP and PM_{10} (Abbey et al., 1995a).

12.5.3 Cancer Incidence

There are few data correlating the occurrence of cancer with chronically high air pollution, specifically with high levels of particulates. Some of the key early studies from the 1950s and 1960s were identified in a review paper by Speizer (1983). The author found that in most of these studies, potential confounders such as smoking and occupational exposure (radiation and asbestos) were not adequately controlled for. These factors were found to have a far greater impact than air pollution on lung cancer rates. The author concluded that the likely effect of air pollution on lung cancer was something greater than zero, but that the estimate was unlikely to exceed 2% of all lung cancers, or 5/100 000 cases in urban males. Thus, the effect on all cancers was considered likely to be less than 1% of all cases. The cancer risk related to general air pollution has been considered to be so low relative to other risks that the time and expense involved in conducting studies large enough to demonstrate the possible low relative risk have not been warranted. In addition to this, levels of pollution have declined significantly over the past 25 years in most parts of the industrial world (Speizer, 1983).

The correlation between air pollution and lung cancer mortality in Harris County (includes Houston), Texas, between 1979 and 1981 was re-examined by Buffler et al. (1988) in an cross-sectional analysis. Previous reports had been published asserting that the high incidence of respiratory cancer among white males in Houston, Texas, provided evidence that exposure to air pollution increased the risk of respiratory cancer. These reports had provided no actual air pollution data or any proper controls for potentially confounding variables, such as smoking, occupation or socio-economic status. After controlling to an extent for smoking status, socioeconomic status and migration for each census tract division (n=249), and including available air pollution data on TSP (but no other air pollutants), the estimated increase in lung cancer deaths for the TSP range of 16 μg/m^3 between the median of the lowest to the median of the highest quartile was 1.9 per 100 000 white males (the total range for TSP was 58.8 to 83.6 μg/m^3). However only 3% of the total variation in lung cancer mortality was explained by TSP. The authors concluded that air pollution in the study area was not a strong determinant of lung cancer mortality and that, unless other risk factors for lung cancer such as smoking could be better controlled for, the hypothesis of an association between air pollution and lung cancer could not be tested.

Lung cancer mortality has occasionally been reported as elevated in daily time-series analyses, a somewhat unexpected association in view of the long latency period required for development of most cancers. For example, in Philadelphia, the RR for dying of lung cancer on the 10% of days with the highest TSP values (mean 141 μg/m^3) was elevated to 1.19 compared to the 10% of days when TSP was lowest (mean 47 μg/m^3) (Schwartz 1994d). This is likely a manifestation of the enhanced effect of particulate and other air pollution on those with preexisting respiratory conditions, rather than any chronic effect.

Mills et al. (1991) examined cancer incidence and mortality in a cohort of 6300 Seventh-Day Adventist non-smokers in California (the AHSMOG cohort) over a six-year period (1977-1982) in relation to concentrations of TSP and ozone. Residence history since 1960, percentage of time spent outdoors and indoors, commuting time, history of exposure to passive smoking, past smoking history, occupational exposure, and history of respiratory disease were determined individually by questionnaire. After adjustment for age, sex, and past smoking history, increased frequencies of exceedance of various cutoffs of TSP from prior exposure (1973-1977) were most closely associated with increases in the risk of all cancer incidences combined among females, for whom the largest increases in risk estimates were observed to occur in the smoking-related respiratory cancers (larynx, lung, pleura). The coefficient associated with mean concentration of TSP (99.7 μg/m^3) increased three-fold from 0.0053 to 0.016 after adjustment for indoor exposure (mean TSP 44.1 μg/m^3). An increased risk was not observed in males. Elevations in cancer risk were observed at TSP concentrations of 100 μg/m^3 for 1000, 2500, or 5000 h/y exposure (42,104, or 208 days) but not at 75 μg/m^3 or 60 μg/m^3 cutoffs. Analysis of cancer mortality (180 deaths in the 10-year mortality follow-up period, 1977-86) revealed somewhat stronger associations in males compared with females. This was somewhat inconsistent with the results for incidence, in which females showed associations while males did not. It was also suggested that TSP may not be the best measure of particulates in determining health effects, and $PM_{2.5}$ was recommended as a possible alternative.

In an updated report (Abbey et al., 1995b) which included TSP, PM_{10}, and $PM_{2.5}$ measurements from 1973 to 1987, the relative risk for incidence of all cancers in females was increased to 1.37 (95% CI 1.05-1.80) for TSP with 42 days/yr>200 μg/m^3. For PM_{10} at 42 days/yr>100 μg/m^3, the RR was marginally increased (RR=1.15; 95% CI 0.97-1.38) This reduced strength of association for PM_{10} could have been due to measurement error, since PM_{10} was estimated from TSP for a portion of that time. Although the RR was doubled for females in association with an average annual increase of 45 μg/m^3 $PM_{2.5}$, the result should be viewed with caution due to the small size of the subcohort for which $PM_{2.5}$ data were available (n=1800), and the consequent wide confidence intervals (RR=2.01; 95% CI 1.05-3.86). The RR for respiratory cancer incidence was also increased for exposure to TSP, PM_{10}, and $PM_{2.5}$, but the increases all included a RR of 1 (Abbey et al., 1995b). These results were based on relatively few cases of newly diagnosed cancers (n=301) and respiratory cancers (n=17) which contributed to the weakness of the findings, especially when subsets were examined.

In a study by Dockery et al. (1993), already reviewed in section 12.5.1 above, the authors examined the association between air pollution and cancer mortality over a 14- to 16-year period in six US cities. After adjustment for age (5 y age groups), sex, education as a surrogate for socioeconomic status, occupational exposure, and smoking status both present and past, the Relative Risks (RRs) for the association between mortality due to lung cancer and particles (fine and inhalable), as well as the other pollutants were slightly elevated, but the confidence intervals were broad. The RR for the association with fine particles was 1.37 (95% CI 0.81-2.31) for a range of 18.6 μg/m^3 , or 19.9% for a 10 μg/m^3 increase in $PM_{2.5}$). Smoking was found to be the factor most strongly associated with mortality due to lung cancer; the RR for current smokers was 8.4 (95% CI 3.0-21.6), six times the overall RR, and the RR for former smokers was 1.9 times the RR for the most versus the least polluted city. Only 8.4% of the total of 143 deaths were due to lung cancer. This probably contributed to the high variance and lack of statistical significance for the increased RR for lung cancer.

The 151-city ACS cohort study (Pope et al., 1995a) also reported an association between sulphates and lung cancer mortality, but not with the more general index of fine particles ($PM_{2.5}$). The total adjusted mortality risk ratio for a range of 19.9 μg/m^3 SO_4^{2-}, adjusted for age,sex, race, cigarette smoke, occupational exposure, education as a surrogate for socioeconomic status, body-mass index, and alcohol use, was 1.36 (95% CI 1.11-1.66), or 18.1% for an increase of 10 μg/m^3 SO_4^{2-}. When the cohort was subdivided by sex and by smoking history, a part of the increase in risk was seen to be due to increases in males, and in never-smokers, although the RR was elevated but non-significant in women and in never-smokers. The results of this study agreed broadly with the results in the much smaller cohort study of Dockery et al. (1993), that increases in fine particle pollution lead to increases in mortality due to lung cancer, but this study found no effect of $PM_{2.5}$ itself, only of the SO_4^{2-} fraction of fine particles. The magnitude of the effect was virtually the same in the Pope analysis for SO_4^{2-} (18.1%) as that in the Dockery analysis for $PM_{2.5}$ (19.9% for an increase of 10 μg/m^3).

12.5.4 Summary of Long-Term or Chronic Exposure Effects on Mortality and Morbidity

In contrast to the large number of studies on daily variations in pollution associated with mortality and morbidity, relatively few studies are available that examine the effects of long-term or chronic exposure on health endpoints. Such exposures, varying between one and 16-20 years duration, were demonstrated to be associated with increases in mortality, lung function, respiratory disease symptoms, and lung cancer in both cross-sectional and prospective cohort studies, which are considered to provide greater weight of evidence than cross-sectional studies. Drawing conclusions regarding community effects from the population-based cross-sectional studies generally has been difficult due to the inability to account for personal demographic and socio-economic factors, all contributing to the probability of confounding of the results. However, several new prospective cohort studies, in each of the three endpoints examined (mortality, respiratory health and lung function, and cancer), have been able to include some of these factors, thus avoiding to some extent at least, the probability of confounding. They have thus been able to shed some additional light on the effects of longer term and/or chronic exposure on health. With respect to exposure, however, all studies (with the partial exception of the Abbey et al. studies (1995a,b)) were still reliant on population-based exposure calculations.

In both of the prospective cohort mortality studies, the probability of survival over a 7 to 16-year period was reduced for people living in the most polluted city compared to the least polluted city, based on the Cox proportional hazards model (Dockery et al., 1993; Pope et al., 1995a). In the six-city study by Dockery and colleagues (1993), the probability of survival over a 14-year period was reduced from approximately 88 to 79%, and represented a four-year shortening of lifespan between the most and the least polluted city, or about two years for the mean pollutant level, based on the graphical presentation of survivorship curves for each of the six cities. An important point illustrated by this analysis is that mortality is not only a matter of "harvesting" or shortening by a few day or weeks the lives of those who are already ill, but that the lifespan is significantly shortened, and moreover, by several years.

In the six-city study, average mortality was increased by 10%, 14% and 32% for each 10 μg/m^3 increase in PM_{15}, $PM_{2.5}$ or SO_4^{2-}, while the mortality increases were much smaller, 6.9% for $PM_{2.5}$ and 7.5% for SO_4^{2-} (10 μg/m^3 increase) in the much larger (Pope et al., 1995a) study. In addition to its much greater geographic coverage (151 cities instead of six cities) and inclusion of many more individuals (552 000 versus 8100), more potentially confounding factors were included. The lower risks calculated in this study are therefore considered to be the more accurate of the two. The two cross-sectional studies also support the particulate-mortality association with a relative risk for increased mortality (10 μg/m^3 increase) of 4.3% for $PM_{2.5}$ and 8.2% for SO_4^{2-} in Ozkaynak and Thurston (1987), and 9.8% for $PM_{2.5}$ and 12.6% for SO_4^{2-} in the cross-sectional study reported in Pope et al. (1995a) which was the companion to the latter authors' cohort study.

Annual variations in particulate air pollution and ozone were associated with reduced lung function in children in a large cohort study in 22 communities in the US and Canada (Raizenne et al., 1996). The annual values were also shown in an accompanying exposure analysis to be representative of the children's chronic exposures over most of their lives (Speizer et al., 1996). Decrements in FVC ranged from 2.4% to 3.5% for the four particle metrics, with particle strong acidity showing the strongest associations. Moreover, acidity was associated with a 2.5 times increase in the proportion of children whose FVC was less than 85% of the predicted value for their age, sex and height. Co-regression with ozone did not alter this proportion, but ozone was shown to have an independent, if smaller association. Other lung function parameters were also affected, but to a lesser extent than FVC. The study results indicate that significant decrements in lung capacity, lung growth, development, and function result from chronic exposure of children to air pollutants. These results contrasted with the results of an earlier cross-sectional study by the same research group on 5400 children in only six cities, in which no effects on lung function were found in association with particulate air pollution (Dockery et al., 1989), probably due to the low power of the earlier study to detect an effects-on-lung-function-as-result of the numbers of observation (six cities).

Respiratory health was also found to be adversely impacted by particulate air pollution in several studies, in both children and adults. The cohort study on over 13 000 children in 24 US and Canadian communities in whom lung function decrements had been demonstrated, also showed that the odds ratio for developing bronchitis in the past year was elevated for communities with high acidity (OR=1.66 for a range of 52 nmol/m^3) and high sulphate levels (OR=1.65 for a range of 7 μg/m^3) (Dockery et al., 1996a). Asthma, wheeze and other asthmatic symptoms, chronic cough and phlegm production were not significantly affected by high particulate levels. There was a suggestion that asthma and prior respiratory disease, as chest illnesses before age 2, may have played some role in elevation of the bronchitis OR, since they were also associated with non-significant increased OR for bronchitis. The findings of increased OR for bronchitis associated with particles were consistent with the previous findings from a smaller cross-sectional study on six cities by the same authors (Dockery et al., 1989), but in that study, PM_{15} was the particle most closely associated with effects on health (no fine particle metrics were available), while in the newer cohort study, acidity, and to a lesser extent, SO_4^{2-} gave the strongest associations. The "asthma effect" was also much more marked in the earlier study, and it was clear that most of the excess bronchitis in the most polluted cities was being reported by children with asthma.

Annual exposure to BS increased the OR for prevalence of phlegm, but not of asthma, wheeze or cough symptoms, in a cross-sectional analysis of a British birth cohort of young adults, aged 23 years (Scarlett et al., 1995). The effect of asthma or other respiratory diseases in childhood on prevalence of symptoms in adulthood was not investigated, although it was apparent that asthma at age 16 had no effect on asthma prevalence seven years later.

The development of new cases of chronic bronchitis was shown to be associated with 10- to 20-year (chronic) exposures to estimated $PM_{2.5}$ in excess of 20 µg/m^3 in a cohort of 1863 non-smoking older adults (average age 48 y) in California who were followed prospectively for 10 years from 1977 to 1987 (Abbey et al., 1995a). Exposures in this cohort were calculated individually, taking into account lifestyles, time-activity, indoor exposure and history of residence and occupation. For a 45 µg/m^3 increase above the mean, the RR for development of chronic bronchitis was 1.8, which is equivalent to a 17.7% increase in incidence per 10 µg/m^3. Relative risks for new cases of airway obstructive disease (AOD) and asthma, although elevated, were not significant because the number of new cases was small. Those who gave some indication of possible pre-existing disease or susceptibility in the first survey were three times as likely to have developed new cases of chronic bronchitis or AOD, and 15 times more likely to have developed asthma. A previous high incidence of childhood respiratory disease was also linked to increased risk of new cases of AOD or chronic bronchitis in these older adults, similar to the findings of Dockery et al. (1989, 1996a) in children. Significant increases over the 10-year period were also recorded in the severity of the symptoms for all three endpoints in association with PM.

Lung cancer appeared also to be associated with increased exposure over long periods to some component(s) of the fine fraction of particulate matter in the two cohort mortality studies previously examined. In the six-city study (Dockery et al., 1993) the mortality incidence of lung cancer over a 16-year period was increased 19.9% for each 10 µg/m^3 increase in $PM_{2.5}$, but the increase was not statistically significant, probably due to the few units of observation in this study. PM_{10} and sulphate results for lung cancer mortality were not presented. The larger cohort study of Pope et al. (1995a) found a similar increase of 18.1% in lung cancer (statistically significant in this case) over a seven year period for each 10 µg/m^3 increase in SO_4^{2-}, but $PM_{2.5}$ was not a significant predictor of cancer mortality in this cohort, in contrast to the six-city results. The remaining cohort, the AHSMOG cohort of Seventh Day Adventists (Mills et al., 1991), gave inconsistent results with respect to lung cancer as related to TSP, possibly because of the use of this coarse particle measurement, and possibly also because of the relatively small size of the cohort combined with the short followup period of six years. Although smoking, past and present, was taken into consideration in all three studies, it exerts such a strong influence on lung cancer rates (for example a relative risk of 8.0 for current smokers in the six-city study) that the possibility of residual confounding cannot be dismissed with respect to the associations seen between particles and lung cancer.

Cross-sectional or prospective cohort studies that examine annual or longer averages in exposure are usually interpreted as indicating the chronic or cumulative effects of exposure. The health consequences of chronic exposure to air pollution, whether the effect is due to cumulative exposures, to some time-window of past exposure, or to residual damage caused by a large number of individual events, have not been as easy to distinguish as acute adverse effects from single day or short-term exposures. It is clear that exposure to air pollution over a period of years results in a substantial reduction of survival, in the order of several years. This reduction cannot be merely a "harvesting" of persons who would have died anyway within a few days or weeks, since the survivorship data are incompatible with such an interpretation. It also seems reasonable that, if highly susceptible persons die on days with elevated pollution levels, there could also be induction of some residual chronic health effect(s) in the survivors. However, the cohort mortality studies do not provide information on this aspect, since either long-term (low) exposure or a long series of acute exposures would be expected to result in the same observed effects on mortality in these studies (Vedal 1996). However, a few additional endpoints strongly suggest chronic toxicity effects. These include the development of new cases (increased incidence) of chronic disease, increasing severity of an adverse effect over time, more rapid loss of function in adults than is the expected case, reduced pulmonary growth rates in children, and lung cancer. Results from studies that have investigated these effects are discussed below.

The development of new symptoms of chronic respiratory disease (such as chronic bronchitis), as noted above, suggests the results of chronic exposure. Many of the time-series studies on acute effects have inferred that symptoms of respiratory disease are manifested entirely in those with pre-existing conditions. Development of new disease in older (mean age 48 y) adults without pre-existing symptoms in response to past chronic exposure was clearly shown in the AHSMOG cohort study (Abbey et al., 1995a). An 18% increase was observed in new cases of chronic bronchitis per 10 μg/m^3 increment in average $PM_{2.5}$ measured for the 10-year period before the study began. The development of airway obstructive disease was also linked to past exposure to TSP and PM_{10} in the four years preceeding the start of the study. Development of chronic bronchitis has also been observed in children, in association with long term exposure to increases in PM_{15} (Dockery et al., 1989), SO_4^{2-}, and particle strong acidity, the latter providing the strongest association (Dockery et al. 1996a). Although measurements were taken for only the year preceding the study, these were shown to be representative of concentrations for the past four to 13 years in the 24-city study (except for PSA, for which only the one year of measurements were available), and thus could be considered to be representative of the children's entire lifetime exposure. Moreover, stratification of the samples by length of residency in the communities showed that life-long reidence was associated with a greater decrement (4.2%) in forced ventilatory capacity (FVC) than residency for only the past three to four years (2.4% decline). It is possible that part or all of the new development of disease was due to pre-existing genetic susceptibility, but the lack of association with parental asthma does not support this hypothesis well. The increased incidence of disease in childhood appears to have additional chronic effects due to carryover of increased susceptibility into adulthood, as observed in the association of increased respiratory illnesses and/or airway obstructive disease as a child with increased odds for development of chronic bronchitis or airway obstructive disease in adulthood (Abbey et al., 1995a).

Over a period of 10 years, increases in severity of respiratory symptoms for airway obstructive disease, chronic bronchitis, and asthma was shown in the Adventists' prospective cohort to be associated with both past and concurrent 10-year average particle concentrations (TSP, PM_{10}, $PM_{2.5}$, and SO_4^{2-}) ; increases in severity of asthma were additionally associated with ozone and visibility (Abbey et al., 1995a).

Decrements in lung function were observed in children in the 24-city study, and increased odds of having a forced vital capacity (FVC) less than 85% of normal were associated with particle strong acidity and ozone (results were not reported for other particle metrics), and the odds were increased by 2.5 times in the most, compared to the least polluted city (Raizenne et al. 1996). This represents a significant decrement in lung growth and capacity, development, and function in children associated with chronic exposure to air pollution. The longitudinal study on lung function declines in adults from one of the six-city analyses demonstrated that age-related lung function declines were accelerated in those who had reported symptoms of chronic cough 12 years previously (Sherman et al., 1982). The Abbey et al. (1995a) results from the Adventist cohort provide a link between these accelerated lung function declines in adults and the occurrence of symptoms of chronic respiratory disease associated with chronic exposure to particles.

Lung cancer is manifested only after a long latency period, and is generally considered to be a multi-step process leading to a chronic condition. In both the prospective cohort mortality studies (six-city and ACS), lung cancer rates in cities with more air pollution were higher than rates in less polluted cities, and were about the same, per 10 μg/m^3 increase, for $PM_{2.5}$ in the six-city study as for SO_4^{2-} in the 151 city ACS study (Dockery et al., 1993; Pope et al., 1995a). Thus the lung cancer results give limited support for a role for particulate matter air pollution in elicitation of chronic effects.

12.6 REVIEW STUDIES, PRIMARILY ON PM_{10}

Because of the large number of recently published studies linking particulate matter concentrations in the environment to observed health effects in the general population, several review papers evaluating the data have been published.

A review of the health effects of particulates was carried out by Withey (1989). The author stated that numerous confounding variables, such as personal habits, weather, climate and lack of definite thresholds, have made correlations between exposure and response, as well as selection of protective or acceptable levels of particulate pollution, very difficult to determine. Recognizing this, the author also recommended that further research should use modern multivariate statistical methods and improved analysis of data to refine and improve the definition of the dose-respose relationship. It was also acknowledged that there existed special sensitive subgroups, such as young children, the elderly and those with COPD. Furthermore, based on some of the data reviewed, it could be concluded that long-term exposure to high levels of particles has been shown to induce or accentuate heart and lung disease (Withey, 1989).

Pope et al. (1995b,c) reviewed approximately 70 references published since 1985, which focussed not only on the health effects of PM_{10}, but also on all measurements of particulates. These consisted of acute and chronic morbidity and mortality studies evaluating particulate levels commonly observed in cities across a wide geographical spectrum and were considered by the author to be competently carried out. Based on evaluation of the data, it is evident that there exists a wide variety of health endpoints associated with particulate levels, including decreased lung function, increased hospitalizations, health care visits for cardiopulmonary disease, increased respiratory morbidity (as measured by absenteeism from work or school or other restrictions in activity) and increased cardiopulmonary disease mortality. Health effects were reported at levels common to a number of both US and Canadian cities, where no threshold could be identified. There appears to be evidence to support a linear dose-response relationship. Future research was recommended, to be directed at finding a biological mechanism to help answer why particles have been so closely linked to these measures of morbidity and mortality.

A review and meta-analysis of 13 studies reporting associations between particle exposure and mortality were carried out by Schwartz (1994e). These studies associated short-term changes in particulate air pollution with short-term changes in daily mortality in their respective study locations. A meta-analysis was conducted by analyzing the measure of the effect and computing an average coefficient, an estimated standard error for that coefficient and often a weighted average. Several criteria were taken into account in assessing these time-series studies, such as potential for confounding by other pollutants, quality of exposure assessment (measurement size and location of monitoring) and quality and quantity of levels of temperature and humidity (weather data) in the cities under study. Results showed that the unweighted meta-analysis of all the studies resulted in a relative risk of 1.06 (95% CI=1.05-1.07) for a 100 $\mu g/m^3$ increase in TSP mass. The most likely sources of potential confounding, such as infectious diseases, weather and other pollutants, were discussed at length with respect to the specific studies. The evidence suggested that it is unlikely that the observed associations resulted from any of the confounders.

A review and analysis of several cross-sectional and time-series studies examining the occurrence of daily fluctuations in air pollution and mortality were conducted by Ostro (1993). For inclusion in the meta-analysis, certain criteria had to be met, such as proper study design and methodology, minimal confounding and omitted variables, concern for the effects of seasonality and weather and acceptably complete analysis of the data. Because of varying measures of particulate matter used in the different studies, general estimates of the relationships between the measurements were used. The authors concluded that the studies generated consistent results. The means of the estimated effect on mortality of a 10 $\mu g/m^3$ change in PM_{10} implied by these studies ranged between increases of 0.31 and 1.49%, with a mean of 0.96%. The means of the lower and upper bound estimates were 0.63% and 1.30%, respectively. Evidence for specificity of effects was also demonstrated, as most of the studies demonstrated an association with disease-specific mortality, such as respiratory or cardiovascular diseases.

Wood smoke has been associated with adverse effects on health and is made up of inhalable particles as well as PAHs, CO and a large group of other compounds, including hydrocarbons and NO_2. Vedal

(1993) and Larson and Koenig (1995) reviewed and summarized evidence relevant to an assessment of the human health effects of wood smoke. The component of wood smoke that is of most significance in causing adverse health effects is fine particulate matter. Exposure to wood smoke has been shown to increase the risk of serious respiratory illness in children and to cause decreases in children's lung function, with asthmatic children probably being more susceptible. Mutagenicity studies such as the Ames bioassay and sister chromatid exchange have identified wood smoke as a mutagen. In the case of combustion emissions, mutagenicity and carcinogenicity seem to be highly correlated, inferring that the carcinogenicity of wood smoke particles is due to their ability to cause mutations. Inhalable particles such as those contained in wood smoke have been associated with a number of health effects, ranging from reduced pulmonary function in children to increased mortality. Levels of particles found in regions of British Columbia that have been attributed to wood smoke may be high enough to cause adverse respiratory health effects. Exposure to wood smoke is estimated to cause substantial morbidity in terms of increased respiratory symptoms and functional limitations. Wood smoke is also thought to cause increases in emergency room visits for asthma, increases in respiratory hospitalizations and excess mortality, although the extent of the impact of these effects on the general population is estimated to be relatively small.

12.7 REVIEW OF FINE PARTICLE EFFECTS

Ambient particulate matter is composed of a heterogeneous mixture of particles varying in size and chemical composition. The attributes that determine the toxicity of particles are very little known. The size distribution of particles is a very important determinant of inhalability and eventual deposition within the respiratory tract. It has been recognized that in order to be inhalable and to reach the tracheobronchial area of the respiratory tract, particles must be smaller than about 10 μm in diameter. Particles 2 to 3 μm and smaller are able to reach the alveoli in the distal parts of the lung, and have been termed "respirable". It thus seems reasonable to assume that these and smaller particles may be more pathogenic than larger particles. Also, particles generated by combustion processes appear to be more pathogenic than airborne dust from crustal origins. The former are mostly in the smaller size fractions, while the latter are generally larger. The chemical composition of particulate matter (for example, sulphates, nitrates, or acids) which varies greatly according to source, has also been hypothesized to play an important role in its toxicity. The number of particles, rather than their mass, has also been suggested as an important determinant of toxicity, since large numbers of very small particles have a very high surface to volume ratio. They therefore present greater opportunities for surface adsorption of toxic substances such as heavy metals or PAHs, and subsequent deposition deep in the lung. Regulatory agencies have therefore begun to turn their attention to the finer inhalable particles, at or below about 2.5 μm in diameter, since strategies to reduce or prevent adverse impacts of air pollution on public health are more likely to be successful if focussed on fine particles of some kind.

A review of the evidence that the fine particle fraction is the critical size range determining the adverse health effects that have been associated in epidemiological studies with increases in airborne particles was commissioned by Health Canada in 1996, and prepared by Dr. S. Vedal, of the University of British Columbia. This section is based partly on that review (Vedal 1996).

The most valuable health studies for the purpose of attributing effects to a specific particle size and composition are those studies in which several particle measures were employed, and particularly if these were not too highly correlated with each other. Typically, when PM_{10} and $PM_{2.5}$ concentrations are both measured, it is often not possible to distinguish the effects of one from another, because $PM_{2.5}$ is a part of PM_{10} and is usually highly correlated ($r \geq 0.6$). However, the coarse fraction of PM_{10} larger than 2.5 μm is often not highly correlated to $PM_{2.5}$ because it stems from different sources than the finer fraction. A few recent carefully conducted studies that included large data sets, have directly compared the coarse fraction of PM_{10} and fine inhalable particles.

Table 12.6 presents data on recent studies on acute and long-term effects on mortality of both fine and coarse particle measures. Because sulphates, which are secondary products of combustion processes, have consistently shown correlation with adverse health endpoints, they have been included in the table along with $PM_{2.5}$. Particle strong acidity, as H^+, has also been suggested, partly on the basis of evidence from animal toxicity studies, as a good candidate agent for the observed health decrements during air pollution episodes.

Table 12.6 Comparison of Fine Particle Effects with Coarse Particle Effects – Mortality

Location (reference)	**Study Type Duration**	**Fine Particles mean (µg/m^3)**	**% Change/10 µg/m^3 increase (95% CI)**	**CP ($PM_{2.5-10}$) (PM_{10}) (µg/m^3)**	**% Change/10 µg/m^3 increase (95% CI)**	**other pollutants, correlations PM interactions**
Effects of acute exposures (daily, several days, or weekly) on mortality						
St Louis, MO, US (Dockery et al., 1992)	time series 1 yr. 1985-86	$PM_{2.5}$ 17.7 SO_4^{2-} 8.0 H^+ 9.7 nmol/m^3	1.5% (0.0 to 3.7%) 6.3% (n.s. t=1.05) 0.9% (n.s. t=0.73)	$PM_{2.5-15}$ (not given) (PM_{10} 27.6)	"effects no different than $PM_{2.5}$" (1.5%, t=2.17) +ve	SO_2 - n.s. NO_2 - n.s. O_3 - n.s. order of significance: $PM_{2.5}$ = $PM_{15-2.5}$ = PM_{10} >SO_4^{2-} > H^+
Kingston-Harriman, TN (Dockery et al., 1992)	time series 1 yr. 1985-86	$PM_{2.5}$ 21.0 SO_4^{2-} 8.7 H^+ 36.1 nmol/m^3	2.3% (n.s. t=1.23) 8.3% (n.s. t=0.67) 0.2% (n.s. t=0.31)	$PM_{2.5-15}$ (not given) (PM_{10} 30)	effects same as $PM_{2.5}$, i.e. n.s. (1.6% , n.s., T=1.07) n.s.	SO_2 - n.s. NO_2 - n.s. O_3 - n.s.
Philadelphia, PA (Dockery et al., 1996b)	time series summers, 1991-92	$PM_{2.5}$ (mean not given) H^+ (mean not given)	2.0% (0.4 to 1.35%) n.s. (p=0.57)	$PM_{2.5-10}$ (not given) (PM_{10} not given)	(p=0.85) n.s. 1.2% (p=0.06) marginal	order of significance: $PM_{2.5}$ > SO_4^{2-} > PM_{10} > H^+ > $PM_{10-2.5}$
6 US cities – overall (Schwartz et al., 1996a; US EPA 1996)	time series 8 yrs., 1979-87	$PM_{2.5}$ 14.7 SO_4^{2-} 5.8 H^+ 10 nmol/m^3	1.5% (1.0 to 1.9%) 2.2% (1.3 to 3.2%) n.s.	$PM_{2.5-10}$ 9.0 (PM_{10} 25.0)	0.4% (-1.0 to +1.0%) n.s. 0.8% (0.5 to 1.0%) +ve	PM correl. high; no coregressions Order of significance (by RR for IQR): $PM_{2.5}$ >PM_{10} >SO_4^{2-} >$PM_{10-2.5}$ =H^+
(6-city) Portage, WI	(see above)	$PM_{2.5}$ 11.2	0.5% (-1.2 to 2.3%)	$PM_{2.5-10}$ 6.6 (PM_{10} 17.8)	0.5% (-1.2 to 2.3%) n.s. -0.7% (-0.4 to 1.7%) n.s.	r=0.32, $PM_{2.5}$-CP
(6-city) Topeka, KN	(see above)	$PM_{2.5}$ 12.2	-1.3% (-3.3 to 0.6%)	$PM_{2.5-10}$ 14.5 (PM_{10} 26.7)	-1.3% (-3.3 to 0.6%) n.s. -0.5% (-2.0 to 0.9%) n.s.	r=0.29, $PM_{2.5}$-CP
(6-city) Boston, MA	(see above)	$PM_{2.5}$ 15.7	2.2% (1.5 to 2.9%)	$PM_{2.5-10}$ 8.8 (PM_{10} 24.5)	0.2% (-0.6 to 1.2%) n.s. 1.2% (0.7 to 1.7%) +ve	r=0.23, $PM_{2.5}$-CP
(6-city) St. Louis, MO	(see above)	$PM_{2.5}$ 18.7	1.1% (0.4 to 1.7%)	$PM_{2.5-10}$ 11.9 (PM_{10} 30.6)	0.2% (-0.7 to 1.1%) n.s. 0.6% (0.1 to 1.0%) +ve	r=0.45, $PM_{2.5}$-CP
(6-city) Knoxville, TN	(see above)	$PM_{2.5}$ 20.8	1.4% (0.2 to 2.6%)	$PM_{2.5-10}$ 11.2 (PM_{10} 32.0)	1.0% (-0.6 to 2.6%) n.s. 0.9% (0.1 to 1.8%) +ve	r=0.44, $PM_{2.5}$-CP
(6-city) Steubenville, OH	(see above)	$PM_{2.5}$ 29.6	1.0% (-0.1 to 2.1%)	$PM_{2.5-10}$ 16.1 (PM_{10} 45.6)	2.4% (0.5 to 4.3%) ve 0.9% (0.1 to 1.6%) +ve	r=0.69, $PM_{2.5}$-CP
Buffalo, NY (Gwynn et al., 1996)	time series 2.5 yrs, May 1988-October 1990	SO_4^{2-} (not given) H^+ (not given)	(~12% for max-mean) (~22% for max-mean)	CP not separated (PM_{10} est. from 6-d monitoring)	— (~12% for max-mean)	O_3, NO_2 ,CO also sign for total mortality, SO_2, n.s. None signif. for resp. mort. Both H^+ and gases associated in multiple regression. Order H^+ > SO_4^{2-} > PM_{10} > CoH

Table 12.6 Comparison of Fine Particle Effects with Coarse Particle Effects – Mortality *(continued)*

Location (reference)	**Study Type Duration**	**Fine Particles mean (μg/m^3)**	**% Change/10 μg/m^3 increase (95% CI)**	**CP ($PM_{2.5-10}$) (PM_{10}) (μg/m^3)**	**% Change/10 μg/m^3 increase (95% CI)**	**other pollutants, correlations PM interactions**
Amsterdam, NL (Verhoeff et al., 1996)	time series 7 yrs, 1986-92	BS 12 (BS are FP, size to 7μ but underest. SO_4^{2-}, NO_3 etc.)	1.9% (0.2 to 3.8%) +ve (1.8% +03 ; 2.7% +SO^2; 2.0% +CO)	— (PM_{10} 38)	— 0.6% (-0.1 to 1.4%) marg	O_3 (lag 2d) 0.5% (.0 to 1%) O_3 RR reduced to 0.3% +BS; O_3RR same + PM_{10}, but both n.s. SO_2, CO n.s. alone or with BS, PM_{10} Order of significance: BS >PM_{10}
Paris, France (Dab et al., 1996)	time series 6 yrs, 1987-92	BS 31.9	0.7% (-0.25 to 1.8%) n.s.	(PM_{13} 50.8)	1.7% (0.4 to 3.1%) +ve	SO_2 (1 h) 0.85% (0.1 - 1.6%) singly O_3 (1 h, 24 h), NO_2 (1 h, 24 n) n.s. no correl. Coeff., no coregressions Order of significance: PM_{10} >BS
Effects of longer term or subchronic exposure on mortality						
6 cities, US (Dockery et al., 1993)	prospective cohort 14-16 yrs follow-up, 1974-91	$PM_{2.5}$ 16.8 (median) SO_4^{2-} 7.3 (") H^+ 15 (") (nmol/m^3) non-SO_4^{2-}- $PM_{2.5}$ (n.g.)	(rate ratio incr., low-high) 14% (3-27%) 35% (7-66%) n.s. 29% (19-38%)	$PM_{15-2.5}$ n.g. (PM_{15} 28.9) TSP 53 (TSP 1970>100)	(increase in rate ratio, low-high cities) 19% (-9 to +57%) n.s. 9% (3 to 17%) +ve 4% (-0.001 to 9%) n.s. 1% (0 to 3%) weak+ve	no co-regressions. Correlation coefficients not shown, but generally high (reason for no coregression) O_3 (24 h) n.s. SO_2, NO_2 significant.; rate ratios (10ppb) =1.10, 1.13 resp. and lower conf. limits 1.00 (both) Order of significance: $PM_{2.5}$-SO_4^{2-} > $PM_{2.5}$>SO_4^{2-}>PM_{15}>TSP >$PM_{10-2.5}$ > H^+
151 metro areas, US (50 for $PM_{2.5}$, 151 for SO_4^{2-}) (Pope et al., 1995a)	prospective cohort 7.3 yrs follow-up, Sept 1982-end 1989	$PM_{2.5}$ 18.2 SO_4^{2-} 11.0	6.6% (3.6 -9.9%) 7.2% (4.4-10.5%)	no other PM measures	—	no consideration of any other PM or gaseous air pollutants Order of significance: SO_4^{2-} >$PM_{2.5}$
50 and 151 metro areas as above, US (Pope et al., 1995a)	cross-sectional 1980 data	$PM_{2.5}$ 18.2 SO_4^{2-} 11.0	9.2% (CI not given) (8.0+1.4 deaths/y/10^5) 11.9% (CI n. given) (10.5+1.3 deaths/y /10^5 per μg/m^3)	no other PM measures	—	no consideration of any other PM or gaseous air pollutants Order of significance: SO_4^{2-} >$PM_{2.5}$
98 metro areas, US (38 areas with FP, IP) Ozkaynak and Thurston (1987)	cross-sectional 1980 data	$PM_{2.5}$ 19.9 (meas.) 23.1 (est.) SO_4^{2-} 11.1	4.3% (on meas. $PM_{2.5}$) (3.7+1.7 deaths/y/10^5) (3.0+1.3 deaths/y/10^5) 8.2% (7.0+1.4 deaths/y/10^5)	no CP separately PM_{15} 37.8(meas.) 47.6 (est.) TSP 77.6	— 1.9% (meas. PM_{15}) (1.6±0.8 deaths/y/10^5) marg (0.7±0.4 deaths/y/10^5) marg 0.35% (0.4±0.3 deaths/y/10^5) ns	When SO4co-regr. with all others, β increased to 7.5 deaths/y/10^5, others n.s. Correl. coeff., SO_4^{2-}-$PM_{2.5}$ high r=0.65, r<0.14 for PM_{10}, TSP Order of significance of regre. coeff. SO_4^{2-} >$PM_{2.5}$ >PM_{15} >TSP

One of the most interesting reports on the relative effects of a wide range of particle metrics comes from the recent extension and re-analysis of the six-city acute mortality data (Schwartz et al., 1996a; US EPA 1996). Coarse particles (CP), as the fraction between $PM_{2.5}$ and PM_{10} in size, were measured and compared directly to PM_{10}, $PM_{2.5}$, SO_4^{2-}, and H^+. While $PM_{2.5}$ (median 14.7 µg/m^3) was associated in the overall results for all six cities with a 1.5% increase (95% CI 1.1 to 1.9%) in total mortality per 10 µg/m^3, coarse particles ($PM_{10-2.5}$) were associated only with a 0.4% increase (95% CI -0.1 to 1.0%). Moreover, when the pattern was examined for each of the six cities individually, CP were significantly associated only in one city, in which a 2.4% increase in mortality per 10 µg/m^3 was predicted. This occurred in the only city in which the correlation coefficient between $PM_{2.5}$ and CP was high, strongly suggesting attribution of the effect from $PM_{2.5}$ to CP. In the other five cities, a -1.3 to a 1.0% increase (Lower CI -0.6 to -3.3%) was predicted per 10 µg/m^3 increase in CP. In contrast, the predicted increases for $PM_{2.5}$ were between -1.3 and 2.2% for the six cities individually, with the results not significant in the two cities having the lowest $PM_{2.5}$ values (11.2 and 12.2 µg/m^3). The order of significance (based on RR for the equivalent interquartile ranges) for all the particle metrics in this study was: $PM_{2.5}$ (2.1%) >PM_{10} (1.8%) >SO_4^{2-} (1.5%) > $PM_{10-2.5}$ (0.4%) ~ H^+ (0.4%).

The three other individual cities (St. Louis, MO, Kingston, TN, Philadelphia, PA) in other studies for which a direct comparison of $PM_{2.5}$ and coarse particles was possible, also indicated that $PM_{2.5}$ was associated with a 1.5 to 2.3% increase in mortality per 10 µg/m^3 increase, while CP was not significantly associated (Dockery et al., 1992, 1996b). The order of significance in the more recent study in Philadelphia was: $PM_{2.5}$ >SO_4^{2-}>PM_{10} >H^+ = CP (Dockery et al., 1996). A similar pattern was shown in a long-term prospective cohort study in the same six cities, in which a 14% rate ratio increase in all-cause mortality was shown to be associated with a 10 µg/m^3 increase in $PM_{2.5}$ (Dockery et al., 1993). The increases in mortality for larger particles (10 µg/m^3 increases) were reduced, at 9% for PM_{15}, 4% for TSP (marginally significant), and a non-significant increase of 19% (95% CI -9 to + 57.%) for CP ($PM_{15-2.5}$). The order of significance of the particle metrics examined in this study was: non-sulphate $PM_{2.5}$ >$PM_{2.5}$ >SO_4^{2-} >PM_{15} >TSP>H^+.

In a cross-sectional study on 98 metropolitan areas in the US, annual variations in two fine ($PM_{2.5}$ and sulphate) and two coarse particle metrics (PM_{15} and TSP), were examined (Ozkaynak et al., 1987). Unfortunately this study did not examine the coarse particles reaction of PM_{15} (i.e., PM_{15}-$PM_{2.5}$). An increase of 4.3% in total mortality per 10 µg/m^3 was associated with $PM_{2.5}$, while the increase associated with PM_{15} was 1.9% and with TSP was 0.35%, both only marginally significant.

In summary, the rate ratio increases for all four of the subchronic exposure studies (Table 12.7) were significant, and varied between 4.3% and 14% for a 10 µg/m^3 increase in $PM_{2.5}$. The highest value was obtained for the six-city cohort study (Dockery et al., 1993), while the remaining cohort study (Pope et al., 1995) and the two cross-sectional studies (Pope et al., 1995a; Ozkaynak and Thurston,1987) gave values that were 34 to 69% below that in the six-city study. This is likely to be due at least in part to the much broader number of cities in different geographic areas with different climatic and socioeconomic regimes that were included in these studies, compared to the six carefully chosen northeastern and central cities in the study by Dockery et al. (1993).

Thus, in almost all cases in both the acute and subchronic mortality studies, fine particles, as $PM_{2.5}$, had a stronger and more significant association with mortality than more coarse particles, as either the particle fraction between $PM_{2.5}$ and $PM_{10/15}$, or PM_{10} and/or TSP. In only two cases was there an association between coarse particles (CP), as $PM_{10/15-2.5}$, and mortality. In the early analysis of the St. Louis data the effects of CP were stated to be "no different than $PM_{2.5}$" but exact figures were not presented (Dockery et al., 1992). The only city of the six-city study in which a positive association was found for CP showed a high correlation coefficient between CP and $PM_{2.5}$ (r=0.69), and in this case the $PM_{2.5}$ regression coefficient was not significant, making it likely that the effect was ascribed in the regression analysis to CP rather than $PM_{2.5}$ (Schwartz et al., 1996a). The strength of the association of mortality with PM_{10} was notably lower than that for $PM_{2.5}$ in both the acute and subchronic studies in all cases except the aforementioned St. Louis results. In both the acute and subchronic six-city studies and in four individual cities (six if the cities comparing BS are included), results were positive, while in Philadelphia (Dockery et al., 1996b), Amsterdam (compared here with BS) and in 38 cities of the

98 city cross-sectional subchronic study (Ozkaknak and Thurston, 1987) results were marginal. The associations for PM_{10} were negative in the first analysis of eastern Tennessee (Dockery et al., 1992) in which a smaller data set was available than in the later six-city study (where it was small but positive), and in Portage and Topeka in the same study (Schwartz et al., 1996a).

Sulphate, which is a part of the fine fraction of particulate matter, was compared with $PM_{2.5}$ in both the acute and subchronic six-city studies (Dockery et al., 1993; Schwartz et al., 1996a), the earlier St. Louis and eastern Tennessee study (Dockery et al., 1992), the prospective cohort study and accompanying cross sectional study on 151 metro areas in the US (Pope et al., 1995a), and the cross-sectional subchronic study on 98 metro areas in the US (Ozkaynak and Thurston, 1987). In the acute six-city study, the association between mortality and SO_4^{2-} was of a lesser magnitude than that for $PM_{2.5}$ for the equivalent IQR. The increase in mortality per IQR increase was estimated to be 1.5 % (95% CI 0.9 to 2.2%) for SO_4^{2-} and 2.1% (95% CI 1.5 to 2.7%) for $PM_{2.5}$ in the acute study (Schwartz et al., 1996a). The increase in the mortality rate ratio in the longer term six-city study (also for a 10 µg/m^3 increase) was 35% for SO_4^{2-} (95% CI 7 to 66%) compared to 14% for $PM_{2.5}$ (95% CI 3 to 27%); Dockery et al., 1993) . In the two cities in the earlier acute study (Dockery and Thurston,1992), the increases for SO_4^{2-} were 6.3% and 8.3%, but they were not significant (t=0.1.05 and t=0.67), probably due to the small number of measurements available (Table 12.7). In the remaining three long-term analyses, SO_4^{2-} was a stronger predictor of mortality than $PM_{2.5}$, and the mortality rate ratio per 10 µg/m^3 increase was 7.2 to 11.9% (Table 12.7), or 9% and 29% higher than $PM_{2.5}$ in the ACS cohort and concurrent cross-sectional analysis (Pope et al., 1995a) and almost twice as high as $PM_{2.5}$ in the cross sectional study on 38 of 98 metro US areas (Ozkaynak et al., 1987). No correlation analyses between PM metrics were presented for any of these studies, but they were likely high (Dockery et al., 1993). Thus, sulphate appears to be in the middle in any of the orders of significance found for the studies in which it was included, unless it was the only fine particle metric, in which case it was at the top.

The question then arises, whether SO_4^{2-} is itself toxic, or whether it is merely a good fine particle surrogate. The only study in this mortality series which provided some information on this question was the long-term cohort study on six cities (Dockery et al., 1993). In the US EPA analysis of this study for the particulate matter criteria document (US EPA 1996), the rate ratio (RR) given for non-sulphate $PM_{2.5}$ was greater than the RR for either $PM_{2.5}$ or SO_4^{2-} alone, the latter being lower than $PM_{2.5}$. This indicates that, under the conditions occurring in this study, the association of the non-sulphate portion of $PM_{2.5}$ with mortality is as strong or stronger than the association of the sulphate fraction, although both show independent associations.

The role of acidity, as hydrogen ion (H^+), was examined in both six-city studies and in four individual cities (Dockery et al., 1992, 1996b; Schwartz et al., 1996a; Gwynn et al., 1996). Acidity was not found to be significantly associated with mortality in five of the six locales and was at or close to the end of the order of significance for the various particle metrics in most of the studies. It was noted that acidity was not significant in eastern Tennessee, which was specifically chosen for inclusion in the study because its average acidity concentration was 36 nmol/m^3, much higher than the average of 10 to 15 nmol/m^3 in the other five cities (Dockery et al., 1992). However, Gwynn et al. (1996, preliminary report) showed that, in Buffalo, NY, an increase from the mean to the maximum acidity was associated with an approximate 22% increase in total mortality, almost twice as much as SO_4^{2-} for its increase from mean to maximum. Respiratory mortality was also significantly increased for H^+, but the results were less robust for respiratory mortality than for total mortality. H^+ had the strongest association of the four particle metrics examined in this study, and was closely followed by SO_4^{2-}, then PM_{10}. CoH, which was also analyzed, was far from significance. Correlation coefficients between SO_4^{2-}, PM_{10}, CoH, and H^+ were noted to be high, but were not presented. The results for H^+ on mortality were coherent with results on total and respiratory hospital admissions, in which paired regressions with the four air pollutant gases were carried out, showing that the association with H^+ was robust to inclusion of these (multiple-pollutant regressions were not conducted for the mortality data). The duration of the study was 2.5 years, and included three summers, during which H^+, O_3, and PM_{10} reach their highest annual levels. No data were available for $PM_{2.5}$, and all comparisons with fine particles were based on SO_4^{2-}.

Tables 12.7 and 12.8 present data on recent studies, mostly time-series analyses, comparing the acute effects of fine and larger particle sizes on hospitalizations and emergency visits to the hospital or to doctors' offices. Two studies examined the effects of a broad range of PM metrics on respiratory admissions in Toronto. Thurston and colleagues (1994) regressed seven PM metrics singly and with O_3, but were unable to co-regress them together because of strong correlations between them. This probably contributed to the mixed picture with respect to the order of significance of the various measurements. Based on p-values, the fine particle metrics $PM_{2.5}$, SO_4^{2-}, and H^+ were more significant predictors of respiratory disease than the coarse particle metrics except PM_{10}, which ranked between $PM_{2.5}$ and H^+. However, when each was co-regressed with O_3, only H^+ remained marginally significant.

Burnett and colleagues, in a very recent study, have again examined the association of respiratory and cardiac disease in Toronto with four fine particle metrics ($PM_{2.5}$, SO_4^{2-}, H^+, and CoH) and two coarse particle metrics ($PM_{15-2.5}$ and PM_{10}), co-regressing each with four gaseous air pollutants individually and together (Burnett et al., 1997). The most robust individual association with increases in both cardiac and respiratory hospital admissions was found with CoH. Individually, the relative risks for respiratory hospitalizations, for an increase equal to the concentration range measured for each pollutant, varied from 1.024 to 1.037 (all significant) (Table 12.8). The association with CoH remained significant after coregression with the gases O_3, NO_2, SO_2, and CO, while the other PM metrics became non-significant. For cardiac disease, the order of significance in individual regressions was CoH >CP >PM_{10} >H^+, with SO_4^{2-} and $PM_{2.5}$ not significantly associated. This suggests that coarse particles may play some role in cardiac disease, if not in respiratory disease. This work also showed the importance of considering the effects of the gaseous pollutants in any conclusions based on regressions with particles. Taken together with what is known of the precursors of PM and the formation of CoH, it also suggests that CoH is acting as a surrogate for air pollution from combustion gases.

All three hospitalization studies (Thurston et al., 1994; Burnett et al., 1997; Delfino et al., 1997) which included $PM_{2.5}$ as one of the fine particles examined, found it to be either first or second in order of significance within the fine particle group, which in general was more significant as a predictor of increases in respiratory hospitalization than the coarse particle group including $PM_{10-2.5}$ (two Toronto studies), PM_{10} (all three studies), TSP and TSP-PM_{10} (Thurston et al., 1994). In the only two studies which included a direct measurement of CP ($PM_{10-2.5}$), CP was last, or second to last in order of significance. Only the even coarser fraction TSP-PM_{10} was less significant in the Thurston et al. (1994) study in Toronto.

Sulphate was examined as one of several fine particle metrics in the three previously cited studies in Toronto and Montréal, and in two studies in Buffalo area (Thurston et al., 1992: Gwynn et al., 1996). In Montréal, SO_4^{2-} was the strongest predictor of hospitalizations compared to $PM_{2.5}$ and H^+, while in the two Toronto studies, it ranked below CoH and $PM_{2.5}$ (Burnett et al., 1997), or below H^+, $PM_{2.5}$, and even PM_{10} in the earlier study by Thurston and colleagues. In the two Buffalo studies, which were both also carried out by this research group, H^+ was more consistently associated with increased hospitalizations than SO_4^{2-}, but the correlation coefficients were very high (r=0.77; Thurston et al., 1992), making it difficult or impossible to distinguish the effects of one from another. These same five studies also allow a comparison of the relative strength of the SO_4^{2-} association compared to coarse particle metrics. In the five comparisons of SO_4^{2-} with $PM_{10-2.5}$ and/or PM_{10}, SO_4^{2-} was clearly the better predictor except in the Montréal study (Delfino et al., 1994). In this case, however, the correlation coefficient between PM_{10} and SO_4^{2-} was 0.90, which suggests that the PM_{10} effect was really due to the fine SO_4^{2-} fraction.

BS had higher risk (with marginal significance) than the larger particle size PM_{13} in Paris (Dab et al., 1996), the only hospitalization study which examined a coarse particle fraction as well as BS. The two studies which examined only PM_{10} largely composed of a coarse fraction have been included in Table 12.9, since they shed some light on the relative non-toxicity of large quantities of the coarse fraction of PM composed of crustal materials (Gordian et al., 1996: Hefflin et al., 1994). This suggests that efforts to control larger particle sizes such as those arising from road dust will be ineffective, or at least much less effective than control efforts directed to smaller, combustion-derived particles.

Table 12.7 Comparison of Fine Particle Effects with Coarse Particle Effects – Hospitalizations and Emergency Department Visits

Location Study Type Period of Study (Reference)	**Endpoints**	**Fine Particles (mean, μg/m^3)**	**% Change/10 μg/m^3 increase (95% CI)**	**Coarse particles CP PM$_{2.5-10}$ (& PM$_{10}$) mean, (μg/m^3)**	**% Change/10 μg/m^3 increase (95% CI)**	**Other pollutants, correlations, PM interactions**
Toronto, Ontario daily time-series 3 summers, July-August 1986-88 (123 d) (Thurston et al., 1994)	Respiratory admissions Asthma admissions	(1986, 1987, 1988) PM$_{2.5}$ (17.7,15.8, 22.3) SO$_4^{2-}$ (74.4, 38.0, 123.6 nmol/m^3) H$^+$ (21., 13, 52 nmol/ m^3)	single pollutant model : respir.(p) asthma (p) 6.5% (0.01) 3.7% (0.08) 0.8% (0.03) 0.7% (0.04) 1.65% (0.01) 2.1% (0.01)	CP (12.7, 13.9, 16.5) PM$_{10}$ (30.4,29.5,38.8) TSP (62, 66, 87) TSP- PM$_{10}$ (32, 35, 47)	single pollutant model respir.(p) asthma (p) 8.9% (0.09) 7.5% (0.12) 4.7% (0.01) 0.4% (0.43) 1.8% (0.07) 0.8% (0.30) 1.3% (0.18) 2.7% (0.07)	Most robust assoc. with O$_3$- sign. with all PM. H$^+$ sign. alone, marg with O$_3$. All other PM fell 50-66% when co-reg + O$_3$ O$_3$ r =0.7 FP; 0.8 SO$_4^{2-}$; 0.5 H$^+$; 0.65 CP; 0.7 IP; 0.6 TSP. Order of sign. (% + p): (resp.) FP> PM$_{10}$> H$^+$> SO$_4^{2-}$>TSP >CP>TSP- PM$_{10}$. (asthma) TSP> H$^+$>TSP- PM$_{10}$ >FP> PM$_{10}$ >CP
Toronto, Ontario daily time-series 3 summers, June-September 1992-94 (388 d) (Burnett et al., 1997)	respiratory admissions cardiac admissions	PM$_{2.5}$ (14; 4-40) SO$_4^{2-}$ (33; 5-229 nmol/m H$^+$ (1; 0-23 nmol/ m^3 CoH (0.8 x103 ft) PM$_{2.5}$ (see above) SO$_4^{2-}$ H$^+$ CoH	RR for range, single regr. respiratory (t); cardiac (t) 1.037 (3.29) 1.031 (1.80) 1.029 (4.07) 1.017 (1.56) 1.024 (3.48) 1.024 (2.30) 1.037 (5.36) 1.062 (5.63) RR, regr. with all gases 0.999 (0.10) 0.993 (0.33) 1.000 (0.02) 0.984 (1.06) 1.006 (0.76) 1.005 (0.46) 1.023 (2.44) 1.059 (3.75)	CP (10; 4-23) PM$_{10}$ (25; 10-58) CP PM$_{10}$	RR for range, single regr. Respiratory (t) cardiac (t) 1.023 (3.41) 1.036 (3.41) 1.030 (3.42) 1.033 (2.24) RR, regr. With all gases 1.007 (0.82) 1.022 (1.68) 1.004 (0.36) 0.996 (0.23)	Order (Single regr., resp) CoH>FP > SO$_4$> PM$_{10}$> H$^+$ ~ CP. (Cardiac):COH >CP > PM$_{10}$ ~ H$^+$>FP(ns)> SO$_4^{2-}$ (n.s.) Order (multiple regr., resp) CoH> all rest (n.s.) (Cardiac) CoH>CP n.s.> all rest n.s. CoH r<0.5 for all exc NO$_2$ O$_3$ signif. In all single and multiple regressions. O$_3$ r< 0.34 for all PM exc. SO$_4^{2-}$ (r=0.53)
Southern, Ontario (incl. Algoma, Sudbury) daily time-series, 6 yrs., 1983-88 (2192 d) (Burnett et al., 1994)	respiratory admissions (May-August calculated)	SO$_4^{2-}$ (5.3) (Toronto 6.7; 95th percentile 20.1)	4.15% (single regr.) 2.08% (Model + O$_3$) 2.05% (model + O$_3$ lag 1, 3 + SO$_4^{2-}$ lag 1 d)	—	—	O$_3$ was more important predictor of resp. admissions than SO$_4^{2-}$ but SO$_4^{2-}$ independently assoc. (P<0.0001) O$_3$- SO$_4^{2-}$ r=0.38-0.65. var. monitors
Southern Ontario (incl. Algoma, Sudbury) daily time-series, 6 yrs., 1983-88 (Burnett et al., 1995)	respiratory admissions cardiac admissions	(PM$_{2.5}$ site-spec. Conversion Factor:15=4.13 SO$_4^{2-}$) SO$_4^{2-}$ (4.37; max 33)	(Adjusted for O$_3$, temp) resp: 0.74% (0.49-0.99%) cardiac: 0.70% (0.36-1.02) resp: 2.7% (1.8-3.6%) Cardiac: 2.5% (1.3-3.7%)	—	—	Site-specific conversion factor available, SO$_4^{2-}$ - PM$_{2.5}$ O$_3$ stronger RR for resp than SO$_4^{2-}$. Correl coeff. O$_3$ – SO$_4^{2-}$ R=0.53 (summer), -0.04 (winter)
Buffalo, NY daily time-series June-August, 2 yrs, 1988-89 (Thurston et al., 1992)	respiratory admissions	— SO$_4^{2-}$ (9.0; max 37.4) H$^+$ (2.2; max 18.7)	9.0% (p<0.01) 28% (p<0.01)	—	—	O$_3$ had highest mean effect. O$_3$ correl. coeff r=0.51, (H$^+$); 0.67 (SO$_4^{2-}$) H$^+$- SO$_4^{2-}$ r=0.77 (can't be disting.)

Table 12.7 Comparison of Fine Particle Effects with Coarse Particle Effects – Hospitalizations and Emergency Department Visits *(continued)*

Location Study Type Period of Study (Reference)	Endpoints	Fine Particles (mean, µg/m³)	% Change/10 µg/m³ increase (95% CI)	Coarse particles CP $PM_{2.5-10}$ (& PM_{10}) mean, (µg/m³)	% Change/10 µg/m³ increase (95% CI)	Other pollutants, correlations, PM interactions
Buffalo, NY daily time-series 3 years, 1988-90 (Gwynn et al., 1996)	total admissions respiratory admissions	— SO_4 (not given) H^+ (" ") CoH (" ")	Total: all sign., RR ≈ 1.02 except CoH (marginal) Resp: (range, mean-max) 20% (14 – 26%) 38% (27 – 51%) 1-2% (not signif.)	— PM_{10} (Not given)	Total: 1-2%(significant) Resp: 18% (12 to 24%)	All gases signif. correlated with resp hospital admiss (t=4 to 7.5 exc CO, t=2) RR reduced only slightly when H^+ coregressed with gases. H^+ most consistent.
Montréal, Québec daily time-series 5 years, 1984-88 (May-Oct, 911 days) (July-Aug, 310 days) (Delfino et al., 1994)	respiratory admissions, asthma and non-asthma	— (May-Oct) (July-Aug) SO_4^{2-} 4.2 4.9 (90th percentile 8.2; 9.7)	— 9.6% for days >8.2 µg/m³, for respiratory non-asthma admissions. (no continuous data given)	— PM_{10} (29.5; May-Oct) (31.5; July-Aug)	2.1% (0.6 to 4.0%) (Asthma)	O_3, temp included in model Correlations high between PM_{10}, SO_4^{2-} (r=0.90); O_3- SO_4^{2-} (r=0.59); PM_{10}- O_3 (r=0.63) Suggested that PM_{10} effect was really FP.
Montréal, Québec daily time-series 2 summers, June-Sept 1992, (66 d) 1993 (98 d) (Delfino et al., 1997)	emergency room visits for respiratory disease	(1992) (1993) $PM_{2.5}$ 18.5 12.2 SO_4^{2-} 51.7 34.8 H^+ 11.3 4.0 (SO_4^{2-}, H^+ in nmol/ m³)	Results for 1992 not sign. single regr. results, 1993 (elderly, > 65 y) 9.6% (1.9 – 17.3%) 18.2% (2.4 -34.3%) (infants, < 2 y) 12.6% (1.0-24%)	— (1992) (1993) PM_{10} 30.1 21.7	(elderly, > 65 y) 7.3% (1.9 to 12.7%)	H^+ signif. only in infants In adults, PM order of signif.: SO_4^{2-} > $PM_{2.5}$> PM_{10} All n.s. when coregr. + O_3.(e.g. 4.5% (n.s.)/10 µg/ m³ for $PM_{2.5}$). (correl. coeffic. v. high; $PM_{2.5}$- SO_4^{2-}- PM_{10} 0.87-0.96; O_3-PM .46-.63 O_3 7.2% for 10 ppb, signif. coregr. with all PM
Anchorage, Alaska daily time-series 22 mo., May 1992-March 1994 (Gordian et al., 1996)	outpatient visits to emergency rooms or doctors' offices, for respiratory disease	— ($PM_{2.5}$ was unusually low fraction of PM_{10}. limited monitoring gave av. ratio 0.26, as low as 0.14	—	— (CP av. 74% of PM_{10}., up to 86%) PM_{10} 48 (37- wkend) (PM_{10} max 600;>100 months after volcano erupt.	— (Results n.s. for 4-mo. period of volcano, 100-600 3.5% (p<0.01) (lag 1d)	CO also examined. Coarse fraction of PM larger than usual; PM sign., but high PM during eruption n.s. for asthma, resp. infections (CP not as important as fine fraction PM_{10}
Eastern Washington St. 3 towns. Daily time-series Oct 1990, all of 1991 (Hefflin et al., 1994)	Emergency Dept. visits for respiratory diseases (various)	— (area subject to blowing dust, therefore $PM_{2.5}$ smaller than average fraction of PM_{10})	—	— (CP high fraction of PM_{10}, due to dust storms) PM_{10} 40; max 1689)	— (bronchitis) 0.35% (p=0.03) OR 1.2 for resp., Oct 1991 (2 dust storms, PM_{10}>1000, vs. Oct 1990)	Although the OR for ER visits was raised slightly in dust storms, this was less than expected on basis of high PM_{10} levels. Concl. that crustal dust n.s.
Paris, France daily time-series 6 y, 1987-92 (Dab et al., 1996)	hospital admissions, respiratory disease COPD, Asthma	BS 31.9; max 268 (39.9 – winter) (24.6 – summer)	0.41% (0.07 – 0.75%)-all -0.05% (n.s.) – COPD 0.43% (n.s.) – asthma	— PM_{13} (50.8; max 138) (54.4 – winter) (47.6 – summer)	— 0.45% (0.04-0.87%) all -0.05% (n.s.) – COPD -0.025% (n.s.) – asthma	No correlations or coregre. BS ≈ PM_{13} (BS CI slightly narrower) O_3 n.s. for any endpoint. SO_2 sign. for all, COPD, asthma. NO_2 sign. for asthma.

Table 12.7 Comparison of Fine Particle Effects with Coarse Particle Effects – Hospitalizations and Emergency Department Visits *(continued)*

Location Study Type Period of Study (Reference)	**Endpoints**	**Fine Particles (mean, μg/m³)**	**% Change/10 μg/m³ increase (95% CI)**	**Coarse particles CP $PM_{2.5-10}$ (& PM_{10}) mean, (μg/m³)**	**% Change/10 μg/m³ increase (95% CI)**	**Other pollutants, correlations, PM interactions**
London, England daily time-series 5 yrs, Apr.1987-Feb 1992 (Ponce de Leon et al., 1996)	hospital admissions, respiratory disease	BS 14.6 (6-27)	RR = 1,0 (n.s. year, both seasons)	—	—	O_3 most signif. assoc. with resp. illness, singly or coregr. with BS, NO_2, SO_2 BS n.s. for all 3 ages, single or double regression
Birmingham, England daily time-series 3 years, 1988-1990 (Walters et al., 1994)	hospital admissions - respiratory - asthma	BS 12.7 (max. 188)	24.6% – resp (lag 2d) 20.9% – asthma (lag 2d)	—	—	SO_2 also assoc. with 17% incr. in asthma (marg.) and 20% incr. in resp.adm., summer + winter (signif. less than BS) No co-regres. No data for other pollutants.
Amsterdam, NL Rotterdam, NL daily time-series 4 years, 1986-89 (Schouten et al., 1996)	hospital admissions - respiratory (all) - COPD - asthma	BS (Amst.) 11 (1-37) BS (Rot.) 26 (6-61)	(A)-RR n.s., any endpoint (R)-3.7% (0.9-1.7%) resp. - n.s. COPD, - n.s. asthma	—	—	(Amst.) O_3 -resp dis positive, marg. (Rot.) O_3 -resp. dis. sign., age 65+ NO_2 sign., resp dis., age 15-64; COPD All ages SO_2 n.s., both cities, all ages
Athens, Greece daily time-series 1 year, 1988 (Pantazopoulou et al., 1995)	emergency visits, - respiratory - cardiac disease	BS 135, 112,75 (winter) 106, 89,55 (summer) (3 monitoring locs.)	respiratory (summer n.s.) 1.6%, 1.1%, 4.1% -winter Cardiac (summer n.s.) 0.8%, 0.9%, 1.5% – winter	—	—	CO also significant, also in winter 1 of 3 monitors, NO_2 in city centre, also signif. in winter. concentration-response poor, possibly because of low no. of events.High collinearity noted (no values given)
Barcelona, Spain daily time-series 5 years, 1985-89 (Sunyer et al., 1993) (Castellsague et al., 1995)	emergency visits for - COPD (Sunyer et al.) - asthma (Castellsague et al.)	BS ~68 (winter) 48 (summer)	COPD: n.s. in summer 2.3% (t=5.0) winter 0.9% (t=2.7) +SO_2 winter asthma: 4.6% single regr., (Lag 0-3d) summer	—	—	SO_2 gave similar increases in winter, also sign., summer, but reduced in coregres. +BS. No other pollutants given SO_4^{2-} was 38% of BS in summer., 23% in winter. RR, SO_2 same + SO_4^{2-}

Table 12.8 Summary of Comparison of Fine and Coarse Particle Effects in Hospitalization Studies

Location	Reference	Fine particles	Coarse particles	Other pollutants	Order of significance
Toronto, ON	Thurston et al., 1994	$PM_{2.5}$, SO_4^{2-}, H^+	CP, PM_{10}, TSP, TSP-PM_{10}	O_3 (significant with all PM in co-regression) Correl. coeff. with PM high, 0.5-0.8.	H^+ most robust (but marginal with O_3). FP>PM_{10}>H^+>SO_4^{2-}>TSP>CP>TSP-PM_{10} For asthma, mixed results
Toronto, ON	Burnett et al., 1997	$PM_{2.5}$, SO_4^{2-}, H^+, COH	CP, PM_{10}	O_3, SO_2, NO_2, CO (O_3 signif. with all PM in pairwise regression; PM n.s. except CoH)	single regression order (respir): CoH>FP>SO_4^{2-}>PM_{10}>H^+≈CP multiple+ gases: CoH>all others Entire effect can be explained by gases without any PM.
Montréal, Qué.	Delfino et al., 1996	$PM_{2.5}$, SO_4^{2-}, H^+	PM_{10}	O_3 (significant in co-regression)	adults: SO_4^{2-}>$PM_{2.5}$>PM_{10}>H^+ infants: H^+> all others (n.s.)
Montréal, Qué.	Delfino et al., 1994	SO_4^{2-}	PM_{10}	O_3 (n.s., + temp included)	PM_{10} > SO_4^{2-} (but r=0.90)
Buffalo, NY	Gwynn et al., 1996	SO_4^{2-}, H^+, CoH	PM_{10}	O_3, SO_2, NO_2, CO	H+>SO_4^{2-}>CoH (marg) PM_{10} µ SO_4^{2-} H^+ gases sign., consistent
Paris, France	Dab et al., 1996	BS	PM_{13}	O_3 (n.s.), SO_2, NO_2 (signif.)	BS ≈ or > PM_{13}
Buffalo, NY	Thurston et al., 1992	SO_4^{2-}, H^+	—	O_3 (more signif. than PM)	H^+ – SO_4^{2-} r=0.77 (can't distinguish apart)
S. Ontario	Burnett et al., 1994	SO_4^{2-}	—	O_3 (>SO_4^{2-})	—
S. Ontario	Burnett et al., 1995	SO_4^{2-}	—	O_3 (>SO_4^{2-})	—
Anchorage, AK	Gordian et al., 1996	—	PM_{10} (74-86% CP)	CO	PM_{10} > CP (CP marginally significant)
E. Washington	Hefflin et al., 1994	—	PM_{10} (high CP from dust)	—	PM_{10} > CP (CP not significant)

Overall, these studies thus support the hypothesis very well that the fine particle fraction is more important as a predictor of toxicity than the coarse fraction. While we do not yet know which components of the air pollution mix are responsible for the toxic effects observed, nor do we know the precise mechanisms by which they act, the consistency of the associations of excess mortality and morbidity with the fine fraction of particulate matter in well-conducted studies supports the need for objectives and other control measures for the fine fraction.

12.8 SUMMARY OF EPIDEMIOLOGY RESULTS

12.8.1 Acute Mortality

Daily or short-term variations in particulate matter, as PM_{10}, BS, $PM_{2.5}$, or sulphate, were significantly associated with increases in all-cause mortality in 43 regressions carried out in 20 cities across North and South America, England, and Europe (18 studies). The magnitude of the relative risk for PM_{10} was small, varying between 0.4% and 1.7% per 10 $\mu g/m^3$ increase, with an unweighted mean of 0.8% and a weighted mean of 0.5% per 10 $\mu g/m^3$ increase. While these increases seem small in terms of relative risk, they signify substantial numbers of avoidable deaths, due to the very large size of the population that is impacted by air pollution.

The magnitude of the increase in relative risk for BS was about the same as that for PM_{10} , and remained the same or slightly higher in two of the three co-regressions with ozone and/or SO_2. In the best-conducted study which examined $PM_{2.5}$, a mean increase in mortality for six US cities of 1.5% per 10 $\mu g/m^3$ was observed; this increase ranged from 0.8 to 2.2% for the six individual cities (Schwartz et al., 1996). The association with $PM_{2.5}$ was stronger than that with PM_{10} in the same study, 1.5% for $PM_{2.5}$ compared to the 0.8% for PM_{10} (per 10 $\mu g/m^3$) (combined results for all six cities). By comparison to other particles, sulphate was also strongly associated with mortality. Although the magnitude of the mortality risk was greater in the six-city study for SO_4^{2-} compared to $PM_{2.5}$, (2.2% compared to 1.5% per 10 $\mu g/m^3$, all cities combined), the strength and robustness of the association was greater for $PM_{2.5}$ than for SO_4^{2-}

These increases in relative risk were observed at ambient concentrations that were usually well below current standards and objectives; the median concentration for PM_{10} in 16 studies from North America in which significant PM_{10}-mortality associations were found was 38 $\mu g/m^3$ (range 24.5-51.0 $\mu g/m^3$). The results were highly consistent under the widely varying climatic exposure conditions and pollutant mixtures encountered in the different locations.

In all of the analyses that examined one (or more) gasous air pollutants together in the same model with particulate matter or which corrected for them in some other way, the association of particulate matter with daily mortality was remarkably robust, despite the problems of disentangling the effects of PM from other air pollutants. Sulphur dioxide was not itself predictive of mortality in most of the analyses in which it was co-regressed with BS or PM_{10}. Both ozone and particulate matter appeared to act as independent factors in the association of air pollution with daily mortality, since their respective regression coefficients were relatively unchanged in bivariate regressions in seven locations. Some evidence was also found for an independent association between CO and mortality, but further studies are required to confirm this. The evidence for an association of NO_2 with mortality was weak in bivariate regressions, but high correlation coefficients between NO_2 and particulate matter could serve to hide any association. The magnitude, robustness and consistency of the PM association across so many locations with differing air pollutant mixtures strongly suggests that PM is the best indicator of the air pollution effect on mortality, and is considered to give some support to PM of some kind as a causal agent in adverse health responses in the community.

12.8.2 Daily Hospitalizations and Emergency Department Visits

All of the 16 studies that examined PM_{10} and one or more respiratory endpoints requiring hospitalization showed significant associations, varying between 0.45% and 4.7% per 10 $\mu g/m^3$ increase in PM_{10} at mean concentrations varying between 25 and 53 $\mu g/m^3$. When regressed together with ozone, PM_{10} remained significant in three locations but became marginal or non-significant in three others in which the correlations between these pollutants was high. In a preliminary study in Toronto (Burnett et al., 1997), five of six particle metrics tested in bivariate regressions with ozone retained their association with increases in respiratory hospitalizations, the exception being SO_4^{2-}, the only measurement which had a high correlation coefficient (r=0.53) with ozone.

In the six studies that undertook bivariate analyses with both PM and SO_2, SO_2 was not clearly shown to have associations that were independent of PM_{10}. While NO_2 was found to be associated with respiratory hospitalizations in several univariate analyses, its independence from PM could not be properly evaluated because of the high correlations between NO_2 and PM in all locations. CO was found to have an independent association with cardiac-related admissions when co-regressed with PM_{10}, (suggesting the possibility that some of the positive PM-total mortality results discussed above could have been due to this seldom-regressed pollutant). Thus, of the five different air pollutants considered in single and bivariate or multiple analyses, particulate matter was the air pollutant with the most consistent and stable association with increases in hospitalizations. Ozone and CO were judged to have independent associations as well as PM.

The respiratory sub-categories chronic obstructive pulmonary disease (COPD) and pneumonia hospitalizations for the elderly ($\geq$ 65 y) were also significantly associated with PM_{10} (2 to 5.7% for COPD and 1.1 to 1.9% for pneumonia were found, both per 10 $\mu g/m^3$ increase in PM_{10}). The increased risk for hospitalizations due to COPD was higher (4.1%) than the overall increase in risk (3.7%) for all respiratory diseases in all age groups, thus confirming that those with pre-existing COPD are a susceptible subgroup. The hospitalization data provided only limited evidence for an association between particulate matter and exacerbations of asthma, although the small number of respondents in many of the studies limited their power to detect an effect. The southern Ontario study (Burnett et al., 1994), with the clearest positive results for asthma, also included exposure to ozone, which has also been linked to asthma. This suggests the possibility that particulate matter might have a relatively small role, if any, in exacerbation of asthma.

Particulate matter was shown to have associations with cardiovascular disease in addition to its associations with respiratory disease, but the magnitude of the cardiovascular associations were generally smaller than those for respiratory disease.

In six of the eight studies which employed BS as the particle metric, the relative risk for respiratory hospitalizations was increased, providing some evidence for an association between increases in BS and hospitalizations for respiratory disease, despite variations observed in strength and significance of the associations and lack of data on its significance when included in regressions with the range of gaseous air pollutants known to occur with it.

Sulphate has a strong association with respiratory hospitalizations, and appears to act as a good surrogate for fine particles in locations where fine particle measurements are not available. This does not mean that sulphate is itself directly toxic. All eight studies which examined SO_4^{2-} found positive associations in univariate regressions, but significance was lost in three of these when SO_4^{2-} was co-regressed with ozone, due to high correlations between them. In the largest and best-conducted study available on SO_4^{2-} (Burnett et al., 1995), a 2.0 to 2.7% increase in respiratory hospitalizations per 10 $\mu g/m^3$ increase in SO_4^{2-} was indicated in regressions which included adjustments for ozone. This was calculated to be equivalent to a 1.1% increase in relative risk per 10 $\mu g/m^3$ increase in $PM_{2.5}$, based on site-specific monitoring and conversion factors.

Particle strong acidity (PSA or H^+) was associated with respiratory hospitalizations in five studies, two in Buffalo, two in Toronto and one in Montréal (Table 12.8). In the Montréal study PSA was associated with respiratory hospitalizations only in infants and not in adults, a finding considered to be spurious by the authors, because of inconsistencies in the association. The association was strong (38% per 10 $nmol/m^3$) in the studies by Thurston and colleagues in Buffalo and Toronto, but high correlations between H^+ and ozone or sulphate meant that the associations could not be attributed to any one of them specifically. When H^+ was regressed with each of the gaseous air pollutants in Toronto in the Burnett et al. (1997) report, significance was lost for H^+.

Although ozone appears to be independently associated with hospitalizations for respiratory disease in analyses of summertime results, adequate studies confirm that the association for particulate matter remains strong, based on bivariate regression results in addition to single regressions. The equivocal results with respect to particulate matter obtained from some co-regressions of the hospitalization data generally reflected high correlations between ozone and particulate matter, making it difficult or impossible to separate out their effects. An alternative explanation is that ozone, and possibly other gases as well, act synergistically with particulate matter to produce adverse effects.

12.8.3 Effects on Respiratory Health

In addition to its effects on increases in mortality and hospitalizations, increases in particulate matter have been shown to cause small reversible decrements in lung function in normal asymptomatic children, and in both adults and children who have some form of pre-existing respiratory condition, particularly asthma. These changes were often accompanied, especially in adults, by increases in symptoms such as chronic cough or bronchitis. Respiratory-related restrictions in activity severe enough to result in an increased number of days lost to work in adult workers and in school absences in children were also demonstrated to be associated with increased concentrations of ambient particulate matter, in some cases PM_{10}, and in others, $PM_{2.5}$ or other fine particles such as SO_4^{2-}.

12.8.4 Long-Term or Chronic Effects

Longer term, subchronic or chronic exposures, varying in duration between one and 16-20 years' exposure, were associated with increases in mortality, respiratory disease symptoms, decrements in lung function and, possibly, with increases in lung cancer in both cross-sectional and more powerful prospective cohort studies. In one of the cohort mortality studies, lifespan was reduced due to increases in particulate matter by an average of two years over a 14-year period (Dockery et al., 1993), an observation incompatible with suggestions that most or all the observed deaths were due to "harvesting", or accelerating death of persons already ill by a few days or a few weeks. Mortality increases associated with a 10 $\mu g/m^3$ annual increase in fine particle concentrations ($PM_{2.5}$ and SO_4^{2-}) were calculated to be about 7% over a period of seven years in a cohort study covering 151 metropolitan areas in the US (Pope et al., 1995a). Significant decreements in lung function (2.4 to 3.5%), lung capacity, growth and development, along with increased relative risks for bronchitis, were shown to result from chronic exposure of children to fine particles, as H^+, SO_4^{2-}, and $PM_{2.5}$, for all or most of their lives.

The development of new symptoms of chronic disease is considered to be an indication of a chronic effect on the system. Development of new cases of chronic bronchitis (18% per 10 $\mu g/m^3$ $PM_{2.5}$) was observed in a cohort of older adults, in association with fine particles measured for the 10 years before the developmental observation period began (Abbey et al., 1995a). Development of new cases of chronic bronchitis has also been observed in children (Dockery et al., 1989, 1996). This increased incidence of disease in childhood appears to convey additional chronic consequences due to carryover of increased susceptibility into adulthood (Abbey et al., 1995a). Increases in the severity of respiratory symptoms over a period of 10 years have been associated with exposure to fine particles from the concurrent 10 years' exposure, and increases in the severity of asthma with exposures from the 10 years preceding the study (Abbey et al., 1995a).

Chronic (or subchronic) exposure to fine particulate air pollution was also shown to be associated with the development of lung cancer, but this association was very small in magnitude by comparison to other lifestyle factors, particularly smoking.

12.8.5 Fine Particles versus Coarse Particles

A few studies have directly compared the coarse fraction of inhalable particles (PM_{10}) with the finer fraction ≤ 2.5 µm and/or with more chemically defined fractions, generally sulphate, but sometimes particle strong acidity (PSA or H^+). The role of the coarse fraction was examined carefully in the recent re-analysis of daily mortality in the six-city study (Schwartz et al., 1996; US EPA 1996). In the overall results for all six cities, coarse particles were not associated with mortality, nor was there any association in any of the individual cities except the only city in which the correlation coefficient between $PM_{2.5}$ and CP was high, strongly suggesting attribution of the $PM_{2.5}$ effect to CP in this case. These results were supported by the chronic results for the same six cities, and by two smaller mortality studies, as well as by one of the two respiratory hospitalization results for Toronto (Thurston et al., 1994).

Crustal or soil-derived coarse particles were not associated with any great increases in respiratory disease in two studies carried out in western North America, despite extremely high concentrations on some occasions (Gordian et al., 1996; Hefflin et al., 1994). This indicates that control of dust, for example from roadways or construction sites, will be less effective in reducing adverse health outcomes than controlling sources of finer sized particles.

In almost all cases in both the acute and longer term studies, fine particles, as $PM_{2.5}$, had a stronger and more significant association with mortality, hospitalizations, and symptoms than measures of coarser particles including $PM_{10-2.5}$ themselves,

PM_{10}, or TSP. Sulphate appeared to have as strong or stronger an association as $PM_{2.5}$ with increased mortality and hospitalizations. In one study in which SO_4^{2-} and the non-SO_4^{2-} fraction of $PM_{2.5}$ were directly compared, the non-SO_4^{2-} portion was equally as, or more toxic than SO_4^{2-} itself, suggesting that SO_4^{2-} would be an inadequate surrogate for all fine particle effects.

BS, despite its smaller size by comparison to PM_{10}, was not always found to be a predictor of either increased mortality or hospitalizations. This has been suggested to be due to its inadequacy as a surrogate for combustion-related and secondarily produced particles from gasoline engines, although it represents the carbon-based soot particles from diesel engines and stationary sources very well. The type of particle pollution that is prevalent in European cities today has changed from soot-based particles to secondarily formed particles from gasoline combustion (Anderson et al., 1996).

The results for acidity have been mixed; some authors have consistently found H^+ to rank highly in associations with respiratory disease endpoints (Thurston et al., 1994; Gwynn et al., 1996) while others have found acidity to rank at or near the bottom (Schwartz et al., 1996; Burnett et al., 1997).

The correlation coefficients between various particle metrics in any one location have generally been sufficiently high to preclude co-regressions, thus making any separation of effects of the various types of particles difficult or impossible.

Overall, the majority of the studies support the hypothesis that it is largely the fine fraction which is involved in exacerabtions of cardiorespiratory disease. There is some suggestion, however, that coarser particles may play some role in cardiovascular disease and in COPD.

13 RISK CHARACTERIZATION

13.1 HEALTH EFFECTS EVIDENCE

13.1.1 Epidemiological Evidence

The data on the health effects of particulate matter have been examined in animal toxicity studies, controlled human exposure studies, and human epidemiology studies. By far the most compelling evidence for adverse health effects of airborne particulate matter at currently experienced levels in the atmosphere has come from the epidemiological studies. These have the advantage of providing indications of the potential for impact on public health of exposure to particulate matter and other air pollution. Many of these studies have used large administrative databases that enhance the objectivity and measurability of the endpoints. Increases in all-cause mortality in 43 regressions carried out in 20 cities across North and South America and Europe were significantly associated with daily or short-term (several days) variations in particulate matter, as PM_{10}. British Smoke Shade or Black Smoke (BS), $PM_{2.5}$, or sulphate (SO_4^{2-}). The magnitude of the mortality risk for PM_{10} was small (in relative terms, but not in absolute numbers of persons affected), varying between 0.4 and 1.7% per 10 µg/m^3 increase in PM_{10}, with a mean of 0.8% and a median also of 0.8% (n=23), for a wide range of mean concentrations (28 to 115 µg/m^3). For the fine fractions, $PM_{2.5}$ and BS, the mean relative risks of death for an increase of 10 µg/m^3 were also elevated, at 1.5% for $PM_{2.5}$ (n=9) and 1.0% for BS (n=6). For the fine sulphate fraction, one study provided an estimate of 2.2% increase in mortality per 10 µg/m^3 increase in SO_4^{2-}.

As was the case with the acute mortality results, increased hospitalizations in almost all studies (n=26) were shown to have significant associations with daily increases in particulate matter of some kind. The relative risk of increases in respiratory hospital admissions for a 10 µg/m^3 increase in PM_{10} was between 0.45 and 4.7% with a median of 1.7% (n=16). These effects were observed at PM_{10} mean concentrations between 25 and 53 µg/m^3. BS, a somewhat smaller particle than PM_{10}, was associated with respiratory hospital admissions in six of eight studies. The relative risk varied from 0.4 to 12.3% for mean BS levels between 12.7 and 75 µg/m^3. Despite the two negative studies, and the few number of studies in which BS was regressed with other co-occurring air pollutants, the weight of evidence suggests some independent association of BS with respiratory disease. Results for directly measured $PM_{2.5}$, available only for Toronto and Montréal in three analyses, demonstrated positive associations with respiratory admissions, with relative risks (univariate analyses) of 2.5 to 9.6% for a 10 µg/m^3 increase in $PM_{2.5}$. Sulphate was associated with respiratory admissions in all eight studies that examined this measure of particulate matter, with increases of 2 to 9% for a 10 µg/m^3 increase in SO_4^{2-}, at mean concentrations between 3.3 and 11.9 µg/m^3. SO_4^{2-} appears to be a good surrogate for fine particles from combustion sources. A 2.7% increase in respiratory admissions per 10 µg/m^3 increase in SO_4^{2-} (co-regressed with ozone) was indicated in southern Ontario in the best conducted study of the series of eight examined (Burnett et al.,1994, 1995). This was calculated to be equivalent to a 1.1% increase (95% CI 0.7 to 1.5%) per 10 µg/m^3 $PM_{2.5}$, based on site-specific monitoring and conversion factors.

Effects on lung function and respiratory symptoms, although much less serious health consequences than hospitalizations or most certainly than mortality, have the potential to have an impact on a much greater fraction of individuals in the population than the effects resulting in hospitalizations or death. Most studies dealt with children; the evidence is clear that both normal and symptomatic or asthmatic children are affected by air pollution, with increases in respiratory symptoms such as cough or wheeze and/or small reductions in lung function in association with short-term increases in particles (Hoek et al., 1990; Johnson et al., 1990; Pope et al., 1991; Vedal et al., 1991; Pope and Dockery 1992; Roemer et al., 1993; Schwartz et al., 1994; Peters et al., 1996). There was some evidence in one study that children with preexisting symptoms of respiratory disease (such as wheezing or chronic coughs) were more adversely affected by air pollution than normal or non-symptomatic children; the symptomatic children had lower lung capacity or function to begin with, and their lung function/capacity was more adversely affected than normal or non-symptomatic children (Pope and Dockery, 1992). Asthmatic adults also were affected by increases in daily or short-term particle levels, with decreases in lung function (Peters et al., 1996) and increases in respiratory symptoms (cough, etc.) (Ostro et al., 1991; Pope et al., 1991; Perry et al., 1993; Peters et al., 1996) mostly in association with fine particles.

Days absent from school in children (Ransom and Pope 1993) and respiratory-related activity restrictions in adults in several US cross-sectional studies (Ostro 1987, 1990; Ostro and Rothschild 1989) have also been found to be increased in periods with high particulate matter air pollution; increases of 2.8 to 16% in reduced activity (for example remaining indoors) were associated with increases of 10 $\mu g/m^3$ in fine particles or SO_4^{2-}. This latter has implications for the economic cost of particulate matter air pollution with respect to the potential for days lost to work.

The results, particularly for mortality and hospitalization data, but also for some lung function and respiratory symptom studies, were highly consistent under differing PM exposure conditions. These associations could not be explained by the influence of weather, season, yearly or daily variations, or other non-pollutant factors.

Most of the mortality and hospitalization analyses examined one or more air pollutants in addition to particulate matter, whose independent association with mortality remained remarkably stable and consistent, despite the problems of disentangling its effects from the other air pollutants SO_2, NO_2, CO, and ozone.

Moreover, in all except a few locations, the magnitude of the PM association was greater than any other air pollutant considered. The independence, consistency, robustness and magnitude of the PM association across so many locations with differing air pollutant mixtures supports the position that particulate matter of some kind is the best indicator of the air pollution effect on mortality, although effects due to (an)other independently acting air pollutant(s), notably ozone, are also likely in some locations.

There is no clear evidence of a "threshold" level for the positive associations between particulate matter and both daily mortality and hospitalization rates. That is, any increase in ambient particulate matter is associated with a statistical increase in mortality and hospitalization rates. While these endpoints have been emphasized because of their ability to provide some measure of quantifiability, they are only the tip of the iceberg with respect to other adverse health effects including exacerbation of respiratory symptoms such as bronchitis, reduced lung function, restricted activity due to illness, loss of workdays or school absences, and increased costs for medication. These particulate matter-adverse health associations are observed at concentrations currently occurring in Canada, which are low by comparison to international standards or to the concentrations observed in pollution episodes in the 1950's and 1960's in which thousands died.

13.1.2 Experimental Human Evidence

Overall, the clinical data do not lend much support to the observations in the epidemiological studies. Relatively few studies are available, and most have been done using acidic aerosols. None of the human clinical studies have used particle generation systems that reflect the complexity of ambient particles. Responses to acidic particle exposures of both normal and asthmatic individuals show considerable intersubject variability in response. Changes in lung function, mucociliary clearance, and airway reactivity have not been seen in normal subjects at ambient concentrations of acidity or acid sulphate or nitrate aerosols. Asthmatic individuals, especially asthmatic children and adolescents, appear responsive to lower concentrations of acidic aerosols, with different threshold concentrations resulting in bronchoconstriction and small decrements in lung function. Conclusive evidence of enhanced responsiveness in the elderly, or in individuals with chronic obstructive pulmonary disease (COPD) is not available. Little evidence of dose-response relationships can be found in the clinical toxicologic literature. The two major reasons for the lack of support by the clinical studies for the findings of the epidemiology studies are the extreme paucity of data on relevant exposures, and the ethical impossibility of investigating effects in precisely those persons most likely to be affected by air pollutants.

13.1.3 Animal Toxicology Evidence

Most animal toxicology studies of particles deposited as aerosols or by the intratracheal instillation route have involved exposures to single materials. Previous particulate matter reviews have focussed on the toxicology of sulphate aerosols. Acidity, particle size, the anionic component and age of the experimental animal were factors determining a bronchoconstrictive effect. The recent evidence reviewed in this document shows effects on the lung attributable to a particle effect, separate from effects related to the composition of the particle.

Acute exposures of laboratory animals to a variety of types of particles, almost always at concentrations well above those occurring in the environment, have been shown to:

- cause decreases in ventilatory function;
- affect mucociliary clearance;
- increase alveolar macrophages and polymorphonuclear leukocytes in the alveoli;
- cause alterations in immunologic responses (particles with known cytotoxic properties, e.g., metals, affect the immune system to a significantly greater degree);
- alter the lung's defence mechanisms against microbial infections (appears to be related to composition and not the particle effect);
- increase or decrease the ability of macrophages to phagocytize particles (related to composition);
- cause a range of histologic, cellular, and biochemical disturbances, including the production of proinflammatory cytokines and other mediators by the lung's alveolar macrophages, (may be related to particle size, with greater effects occurring with ultrafine particles);
- cause increases in mortality (related to chemical composition and to the susceptibility of the host animal).

The most likely particle types to induce acute adverse effects include metals, organics, acids, and acidic sulphates of the fine particle mode, possibly occurring as coatings on fine or even ultrafine carrier particles.

Subchronic and chronic exposures to some types of particles at mass concentrations > 1mg/m^3 result in significant compromises in various lung functions similar to those seen in the acute studies, including in addition:

- reductions in lung clearance;
- induction of histopathologic and cytologic changes (regardless of particle type, mass concentration, duration of exposure or species examined);
- production of chronic alveolitis and fibrosis.

Evidence for particle-induced pulmonary carcinogenicity in the animal studies indicates that high levels of essentially any particle type will cause lung tumours in rats, although the particle effect is very unspecific.

The interpretation of results from experimental particle inhalation studies in animals and their significance for human exposures involves considerable uncertainties. These uncertainties relate to dosimetry of the respiratory tract, differences in the sensitivities of specific target cells, differences in cell populations in the individual airway generations of animal species, differences in metabolic activity of lung cells, and differences in the lifespan between laboratory animals and humans. Therefore, the animal studies contribute primarily to an understanding of the mechanisms which lead to particle effects in humans rather than to quantitative estimates of human health risks.

13.2 POPULATIONS AT RISK

It has been hypothesized that the observed association of PM with adverse health effects on a population basis, may be due to exacerbation of pre-existing conditions, or it may be an enhanced response of a sub-population of sensitive individuals. The epidemiology data appropriately identifies the same susceptible population as would be expected clinically, however, no exposure patterns for these susceptible groups are available. While many studies have investigated the status of persons with asthma in relation to air pollution exposure, surprisingly few have addressed the other groups. Much of the evidence on particulate exposure is based on studies of community samples, particularly children who can be readily reached through schools. It should be noted that designing either human clinical studies or animal studies to address the issue of exacerbation of disease states, is very difficult.

13.2.1 The Elderly

The relative risk of increased mortality due to air pollution has been shown to be elevated for the elderly as compared to younger adults. In Santiago, the RR for total mortality was increased from 1.07 for adults <65 years, to 1.10 for those >65 y, or an age-related increase of about 3% per 100 µg/m^3 PM$_{10}$ (Ostro et al., 1996). In the re-analysis of the Philadelphia TSP data by Samet et al. (1995), the association between mortality and TSP was not significant for persons <65 years, but was for those over 65.

This finding replicates the original findings of Schwartz and Dockery (1992) who showed that the RR rose to 1.095 for the elderly ≥ 65 years, compared to 1.027 (only marginally significant) for those less than 65 years (Schwartz and Dockery 1992). Schwartz (1994) presents a very clear analysis of the relationship between age and untimely mortality associated with particle exposure by comparing the impacts of the London fog episode in December 1952 to the findings from Philadelphia. Daily pollutant levels were about ten times lower in Philadelphia than during the London fog episode. The results were qualitatively similar for the elderly. Relative risks in London were significantly elevated for all 10-year age groups above 35, while in Philadelphia the RR was significant only for the two age groups 65-74y and 75+ years. However, in Philadelphia, a marginal elevation in RR began to be evident for age groups 45+ and 55+. A recent study examining mortality and both PM_{10} and BS in Amsterdam showed an increase in RR spanning 1.19 for adults < 65 years to 1.26 for those ≥ 65 years, using the BS metric. Increased risk of mortality was not so evident using the PM_{10} metric, due to reduced availability of ambient PM_{10} data points (Verhoeff et al. 1996). One of the only analyses that found no apparent effect of particulate air pollution (as BS) on mortality in the elderly was that of Sunyer et al. (1996) in Barcelona Spain, although some effect was observed for the gaseous pollutants NO_2 and O_3. Although the percentage of excess deaths for total and cardiovascular mortality was lower for the elderly ≥ 75 years than for younger persons, it was slightly higher for respiratory mortality in Athens (Katsouyannis et al., 1990).

13.2.2 Infants and Children

The London fog episodes of particulate and sulphur oxide pollution did not indicate that children other than very young infants ≤ 1 year were at higher risk of mortality than other age groups in the population, although the pollution levels were very much above what would be encountered today. In comparing the mortality experience of Philadelphia to London, Schwartz (1994) did not find a comparable rise in mortality for the under one-year age group, but he did note a significant elevation for 5-12 year olds, which was ascribed to increased outdoor exposure for this age group in Philadelphia. Young children were not found to have an elevated risk for respiratory mortality associated with exposure to PM_{10} in Sao Paulo Brazil, although an elevated risk was observed in the elderly in the same city. On the other hand, studies on less serious outcomes including hospitalizations, doctors' visits, lung function, and respiratory symptoms suggest that younger children may constitute a sensitive subpopulation (Raizenne et al., 1989, 1996). Adolescent asthmatics were found to be more sensitive than adult asthmatics to acid aerosols, and to respond to concentrations of acid aerosols that were an order of magnitude lower than those causing a response in normal subjects (Koenig et al., 1989). It is possible that children are more susceptible to particulate matter air pollution because of their activity patterns which often include more outdoor exposure than would be the case for adults.

13.2.3 People with Pre-existing Respiratory or Cardiovascular Disease

People with asthma and COPD have long been considered particularly susceptible to air pollution exposure. Unfortunately, the effects of exposure to particles, and other environmental pollutants, on respiratory diseases such as asthma, are not well understood. The increased airways responsiveness of the asthmatic enhances responses to environmental pollutants, in contrast to the non-asthmatic without increased responsiveness. It is possible that inhalation of allergenic particles (pollen, fungal spores, etc.) may lead to hypersensitivity reactions expressed as allergic rhinitis or asthma. In controlled exposure studies, adult asthmatics have not generally been shown to respond to particulate air pollution with reductions in lung function and increased respiratory symptoms unless concentrations were above about 200 $\mu g/m^3$ (Utell et al., 1983). However, adolescent asthmatics are more sensitive than adults, responding in clinical studies at concentrations as low as 68 $\mu g/m^3$, in the same range as ambient concentrations (Koenig et al., 1989). Although the mortality and hospitalization studies are not very informative on who is at increased risk, some acute and the long-term panel and cohort studies confirm that asthmatics are more susceptible to particulate pollution than normal individuals (Dockery et al., 1989; Abbey et al., 1996).

Animal models of pre-existing lung disease indicate that particle deposition is reduced with chronic bronchitis, emphysema and fibrosis. Recent work on animal models with pre-existing disease showed that in bronchitic rats exposed for three days to concentrated air pollution (at 250-270 $\mu g/m^3$), mortality was observed in 37%, compared to no mortality in control normal rats exposed to those concentrations.

Pathological findings in the bronchitic rats included airway inflammation, bronchoconstriction, pulmonary vascular congestion, and a doubling of pulmonary neutrophils (Godleski et al., 1996). This is one of the first examples of an animal model that could explain the rapid mortality associated with increased particulate matter seen in many epidemiology studies.

13.3 THE WEIGHT OF EVIDENCE REGARDING THE ROLE OF PM

The best evidence that particulate air pollution is causally associated with cardiorespiratory illness is provided by the mass of epidemiological data. These point to a "pyramid of effects" headed by increases in mortality and in hospitalizations for cardiorespiratory diseases, and progressing downward to decreases in lung function in children and in asthmatic adults, increases in respiratory symptoms that can lead to increases in respiratory-related activity restrictions and days lost from work or school, and long-term or chronic effects including reduced survival, reduced lung function and capacity in children, and increases in development of chronic bronchitis and asthma in some adults.

These studies were conducted under a broad range of environmental conditions in many cities on three continents, by a number of different investigators. They nonetheless displayed a remarkable degree of consistency of effects and coherence between different endpoints.

Although the epidemiology studies are observational and population-based studies rather than experimental, and as such do not provide mechanistic evidence, they have been weighted more heavily than the animal toxicology or controlled human chamber studies for several reasons:

- they are the most direct way of assessing the adverse health outcomes of "real world" complex mixtures of pollutants to which people are exposed;
- human populations, unlike laboratory animals, are highly heterogeneous, including individuals who encompass a large range of susceptibilities, disease status and exposures, and whose responses cannot be predicted from classical toxicology studies;
- population studies based on large administrative databases (for example the hospital admissions study in southern Ontario based on a population of 8.7 million people) can demonstrate the impacts of pollution on public health, and possibly even to enable some partial estimate of the costs to society;
- no extrapolation is necessary when assessing the effects on public health of a particular concentration of air pollutant or of an ambient air objective, as measured by the ambient monitoring network, despite our lack of knowledge about the exposure of each individual in the population. We need only know that the correlation is reasonable between the ambient monitor and the personal exposure.

In evaluating the epidemiology studies as a whole, many issues arise, key among them being that of causality. While it is generally accepted that statistical associations drawn from well-conducted randomized experimental studies (animal or human) provide the strongest evidence for causal relationships, little evidence is available from non-epidemiology studies to support or refute a causal relationship based on the associations observed in the epidemiology studies. Observational study designs (case-control study, cross-sectional study or survey, ecologic study and the cohort study) are used to investigate the consequences of exposure in epidemiology studies. Associations found in a particular study may reflect chance, bias, or cause; rarely does a single study provide evidence of an association that is sufficiently compelling to conclude that the association is causal. Uncontrolled bias is a frequently invoked explanation for associations found between air pollution exposure and health. The findings of epidemiologic studies of air pollution and health need to be interpreted within the context set by our understanding of mechanisms of disease pathogenesis, relevant toxicologic evidence, and the findings of other epidemiologic studies.

Some uncertainties in the evidence of a causal relationship in the observed association between adverse health impacts and increases in airborne particles include:

- lack of an accepted biologically demonstrated mechanism;
- lack of quantitative support from experimental animal and/or human clinical studies;
- difficulty in discerning what, exactly, is the toxic moiety;
- confounding, and difficulty of separating out the effects of other co-occurring pollutants;
- problems of misclassification of personal exposure to ambient particles;

- dependence on ecological or community-based exposures, including analytical studies;
- uncertainty regarding the appropriate pollutants to include in statistical modelling.

The lack of an unaccepted biological mechanism for particle-induced effects is on the way to being resolved (see later sections of this Chapter), particularly since there is good biological plausibility for the observed association. The limited quantitative animal and/or human experimental data is also being resolved through the search for demonstrated mechanisms of action, especially in the animal studies. With respect to the clinical studies, more work is needed on actual air pollutant mixtures with the gases. However, clinical studies may never be able to provide complete answers due to the moral and ethical barriers that must remain (see Section 13.1.2).

The problem of deciphering which of the many types of particles is responsible for the associated adverse health effects is a difficult one, due to several factors, not least of which are the high correlations between different particle metrics. Within the past few years, several large, well-conducted studies have been published that support the hypothesis that some form of fine particle metric is more closely associated with respiratory illnesses than the larger particles. However, the larger particles have not been eliminated from consideration, particularly for associations with certain endpoints such as cardiovascular disease. The difficulties with establishing which particle(s) are responsible may be caused by the nature of the etiological agent(s). In the end, it may prove to be several particle metrics, or even that particles do not act alone but in concert with other pollutant gases.

One of the most difficult questions has been, and continues to be, the role played in the toxicity of particulate matter by other gaseous pollutants. Many available studies simply did not, or could not, consider several of these co-occurring gaseous pollutants. Moreover, they are often highly correlated with particulate matter which makes separation of the effects virtually impossible in many locations. Only one or two of all the available mortality and hospitalization studies examined all four gaseous criteria air pollutants. On the other hand, the consistencies of the associations in such a large number of studies in different locations with a variety of pollutant mixtures give some degree of assurance that the relationships have not been confounded by the gaseous pollutants.

Misclassification of personal exposure is of concern, although not a serious obstacle, in studies of air pollution and health. Epidemiological studies often rely on a single or several fixed ambient monitors to characterize the pollution level in a given community. Personal exposures to particles have only been weakly associated with outdoor concentrations in some studies. Individual personal exposures are not known, therefore, there is no direct link between individual exposure and individual outcomes (i.e., exposure and effect). However, the epidemiological data considered in this review is observational and not experimental in nature, that is, exposures are based on populations, not individuals. Reliance on area monitors may be an advantage because, as explained in Chapter six, ambient fine particles measured at a central site can serve as an indicator of population exposure. Fine particles < 1 to 2.5 μm are fairly uniform across an urban area and have a slower rate of deposition that leads to more homogeneity. They also penetrate indoors more readily than coarse particles. Consequently, the associations between fixed monitoring measurements and health outcomes on a population basis may be reflecting a fine particle effect. Therefore, on a population basis, the adverse health effects are associated with concentrations measured at the central site ambient monitors. Personal exposure is not misclassified; the personal exposure data is lacking, though error in exposure estimates generally leads to an underestimation both of risks and of their statistical significance.

The criteria first proposed by Bradford Hill (1965) and modified by succeeding epidemiologists have been used here to provide a framework for considering the epidemiological data with respect to possible causality of association between particulate matter and adverse health.

Probability *(demonstration of a statistical relationship)*

Recent concerns about adverse effects of particulate air pollution on health largely reflect the publication, since 1990, of a large series of analyses based in part on ecologic analyses of routinely collected administrative data. Results show positive associations, often highly statistically significant, between daily mortality rates in urban areas and various measures of particle concentrations, including TSP, PM_{10}, $PM_{2.5}$, BS, and SO_4^{2-}. Various measures of morbidity, including hospitalizations, lung function decrements, increases in symptoms such as chronic cough, restricted activity, days of work lost and school absences have also demonstrated associations with high ambient particulate matter, in some cases PM_{10}, and in others $PM_{2.5}$, or other fine particle measures (BS or SO_4^{2-}). This health evidence has been more extensively reviewed in Chapter 12.

Strength of Association *(relative risks; weak relationships are susceptible to confounding and may reflect a poor measure of exposure or outcome)*

Estimates of the effect on mortality (per 10 $\mu g/m^3$ rise in PM) are on the order of 0.4 to 1.7% increase for PM_{10}, mean 1.5% increase for $PM_{2.5}$, and 2.2% increase for SO_4^{2-}. The effect on hospitalization for one or more respiratory endpoints varied between 0.45 and 4.7% per 10 $\mu g/m^3$ increase in PM_{10}. The respiratory sub-categories' COPD and pneumonia hospitalizations for the elderly ($\geq$ 65 years) were also significantly associated with PM_{10} (2 to 5.7% for COPD and 1.1 to 1.9% for pneumonia, both per 10 $\mu g/m^3$). The increased risk for hospitalizations due to COPD was higher (4.0%) than the overall increase in risk (3.7%) for all respiratory diseases in all age groups. Sulphate has a strong association with respiratory hospitalizations, on the order of 2.0 to 2.7% increase which is equivalent to a 0.7 to 1.1% increase per 10 $\mu g/m^3$ increase in $PM_{2.5}$. As expected, $PM_{2.5}$ and SO_4^{2-} appear to be more potent than PM_{10} in inducing mortality and morbidity. Particulate matter was shown to have associations with cardiovascular disease hospitalizations but the magnitude of the associations was generally smaller than those for respiratory-related hospitalizations. Exacerbation of asthma, increases in respiratory symptoms, and lung function decrements tended to be slightly larger.

Although the magnitudes of these estimates, or increased risks, are seemingly small and unimportant, they represent large numbers of people, since most of the population is exposed, and serious public health impacts.

Consistency *(Has the association been repeatedly observed by different persons, in different places, circumstances and times?)*

Mortality: The results of these studies have been remarkably consistent in indicating a positive association between particulate air pollution and daily mortality. These associations were seen in 43 analyses in 20 cities across North America, South America, England and Europe. Additional sites found these relationships with the particle metric TSP, not considered here. The US cities included in the analyses range from large metropolitan areas, e.g., New York City and Philadelphia, to smaller cities with polluting industry, e.g., Steubenville and Utah Valley. The estimates of the effects have been similar, in spite of the problems of misclassification of personal exposure to ambient particles, and different health care systems. While most of the studies used the time-series analysis, widely differing applications were employed, with similar results.

Morbidity: The animal studies clearly show effects on the lung resulting from the inhalation of particulate matter, effects that can be attributed to a particle effect rather than to the inherent chemical toxicity of the particle. The epidemiology research has used diverse study designs to examine the association between morbidity and PM, including time-series studies, cross-sectional studies, short-term cohort or "panel" studies, and longer-term cohort studies. The array of health outcomes considered in these studies is equally diverse. Significant associations for particulate matter and respiratory hospitalizations were demonstrated most studies examined. Regression modelling indicated adverse effects of particle exposure on lung function level in children and adults, increased respiratory symptoms and medication use, restricted activity days, and increased frequency of reported chronic respiratory disease (Ostro and Rothschild, 1989; Schwartz, 1993). Consistent associations between fine particle air pollution, as SO_4^{2-}, and increased hospital admissions due to cardiovascular diseases have also been reported in Canada and in the US.

Thus, the epidemiological studies do provide evidence of a consistent association between exposure to particulate matter and several of these critical health outcomes.

Specificity of effect *(precision of the association between particles and adverse effects – does X lead only to Y)*

Evidence shows that particulate air pollution-related increases in both mortality and hospitalizations are associated with respiratory effects. Asthma, COPD, and pneumonia or other upper- and lower-respiratory infections are sub-categories of respiratory disease that also have positive associations with particles. Some investigators included non-respiratory categories in their analyses as controls, and found no positive associations. Several studies also found associations with cardiovascular disease (both mortality and hospitalizations) although of weaker magnitude than the respiratory-related associations. Overall, the evidence is considered strong regarding the specifity of effect to respiratory and cardiac outcomes.

Specificity of cause *(– does only X lead to Y)*

With respect to specificity of the agent, the evidence for particulate matter as the causal agent, as opposed to certain gaseous pollutants, is strong in the majority of studies. Although the associations have been observed in a wide range of locations, with differing mixtures of air pollutants, the relationship between PM exposure and effect has been quite consistently positive. The evidence is further discussed below in the section on confounding. The question of which particle metric remains unsettled, but current evidence suggests that some form of fine particles, less than or equal in size to 2.5 µm, is the best predictor of adverse health outcomes.

Temporal Relationship *(Is the exposure followed by the effect?)*

Most investigators have examined the lag time between pollution peaks and onset of effects, some even investigating "reverse lag", or several days prior to the episode to ensure that the time sequence was correct. Daily peaks of particulate matter were followed within 24 hours to several days by untimely mortality, hospitalizations, lung function changes and respiratory symptoms. Thus, the time pattern of exposure and effect adds to the coherence of the picture, except for the rapidity of the effects on mortality.

The lag period for mortality was surprisingly short, being less than 24 hours in a number of studies. This has created some problems in trying to explain what mechanism could be responsible for these sudden deaths, since not enough time would have elapsed for sufficient tissue damage to account for mortality to occur, nor for infections to have progressed to this stage so rapidly unless there were an acute coronary artery spasm and subsequent massive myocardial infarction (see Chapter ten).

Concentration – Response Relationship *(observation of a gradient of risk associated with the degree of exposure)*

Lack of an observed threshold, with responses increasing monotonically from very low ambient concentrations up to much higher levels, was observed with remarkable consistency in many epidemiology studies on acute and chronic mortality and hospitalizations. While many of these data sets have been analyzed with regression techniques that assume a linear relationship between the response variable and the predictor variable, a number of the newer studies have been analyzed using nonparametric models that do not assume any particular shape for the dose-response curve. In addition, the sensitivity of the concentration-response relationship in the epidemiological data to alternative analytical techniques has been explored in a number of the newer studies. Results have been shown to be robust to alternative methods of analysis.

The concentration-response curves for mortality and morbidity versus PM_{10} and the fine fraction particle ($PM_{2.5}$ and SO_4^{2-}) concentrations appear to be linear in the majority of mortality and hospitalization analyses in many locations in the US and Brazil (Dockery et al., 1992; Pope et al., 1992; Saldiva, 1995; Schwartz et al., 1996). This linear relationship is supported by American studies using TSP as the metric (Schwartz, 1991; Schwartz and Dockery, 1992a; Schwartz and Dockery, 1992b). Nonparametric smoothing techniques have been applied to data from several of these locations, and have generally confirmed the approximately monotonic concentration-response relationship, at least in the lower exposure range (Schwartz, 1994a; Samet et al., 1995; Schwartz, 1995; Pope and Kalkstein, 1996; Schwartz, 1996; Burnett et al., 1997).

The linear concentration-response model represents only one biologically plausible representation of the relationship between particle exposure and health risk. Alternative models, e.g., curvilinear and threshold, have also been suggested by the data in several studies. In several European locations that included PM_{10} and $PM_{2.5}$ metrics, the concentration-response curve was curvilinear, with a steeper, linear component at lower concentrations and a slight flattening (lower slope) at very high concentrations. This curvilinear response was seen in data from Koln (TSP and PM_7) (Spix et al., 1996), from Erfurt, Germany for undefined large suspended particulate matter (Spix et al., 1993), from Amsterdam, NL for both BS and PM_{10}, (Verhoeff et al., 1996), and from Athens, Greece for BS (Touloumi et al., 1994).

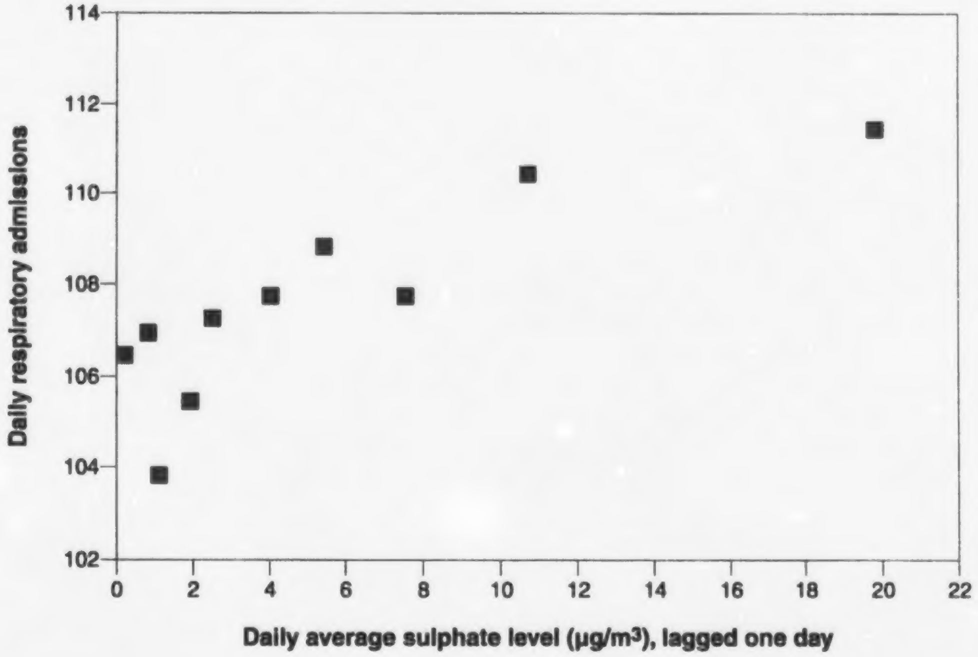
The hospitalization data present a similar picture to the mortality data with respect to the shape of the concentration-response curve. One of the best examples is provided in the study by Burnett et al. (1995) on the association between sulphates and respiratory hospitalizations in southern Ontario (Figure 13.1). The decile curve appears to be slightly curvilinear, and rises monotonically from zero to 20 μg/m^3 of sulphate (lagged one day) without any evidence of a threshold, with a slightly reduced slope at the higher concentrations above about 8-10 μg/m^3. This contrasts with the more obvious linear ozone response in the same study. A threshold for PM_{10} effects was suggested in a re-analysis (by quintiles) and extension of the Utah Valley data by two years (Lyon et al., 1995). The data suggested a 24 hour threshold between 36 and 46 μg/m^3, while the authors suggested 50 μg/m^3. Several other quintile analyses could also be construed as providing support for a weak threshold, due to the low, non-significant relative risks at the lower quartiles or quintiles (Schwartz, 1991; Dockery et al., 1992; Schwartz and Dockery, 1992a,b; Spix et al., 1996). However, overall the data provide much more support for a non-threshold linear or curvilinear response.

In contrast to the epidemiological results, little evidence for a dose-response relationship has been found in the experimental literature despite the fact that the range of particle concentrations used in the human clinical studies usually exceed population exposures associated with the ambient environment. Even at high particle concentrations, acidic aerosols have been found to produce only small decrements in lung function in susceptible subpopulations.

Biological plausibility and possible mechanisms of action

The interpretation of data indicating increased mortality with increased particle concentrations is complex. Studies of extreme particulate air pollution episodes (e.g., the London fog in the 1950s and 1960s) present evidence that high levels of particulate matter are directly associated with increased mortality in the population. The cause of death of these individuals was evident and the role of particles in contributing to the death readily confirmed.

Figure 13-1 Average Number of Adjusted Respiratory Admissions Among All 168 Hospitals by Decile of the Daily Average Sulfate Level (μg/m^3), 1 day Lag.



Source: Burnett et al., (1995).

When evaluating the effects of low level ambient particles, we need to clearly separate acute adverse effects, i.e., mortality and morbidity correlated with daily changes in ambient levels, from chronic effects that reflect long-term exposure to low levels of particulate pollution. Establishing a mechanism for mortality associated with daily variations in particulate air pollution is difficult because of the very short lag period, or in some cases no lag period, between recording elevated particle concentrations and the occurrence of death.

The most clinically relevant links between particulate air pollution and mortality probably relate to exacerbation of severe pre-existing cardiac or respiratory disease, most likely including (1) exacerbation of severe asthma or COPD; (2) increased susceptibility or progression of an acute respiratory infection; (3) worsening of pulmonary edema, due perhaps to either a permeability defect or left ventricular dysfunction; and (4) malignant cardiac arrhythmias. Other hypothesis include "premature" death or harvesting or acute oxidative stress.

In an animal model with bronchitic rats, the bronchitic animals were shown to be extremely sensitive to low doses of freshly concentrated ambient particles by comparison to healthy animals, and died after a few days' exposure. This model of pre-existing disease fits the hypothesis that particulate air pollution, even at low ambient levels, is affecting individuals who have already compromised health. However, in considering the mechanisms by which low concentrations of inhaled particles could lead to mortality, there are uncertainties.

Particle dosimetry in the respiratory tract is an excellent starting point for discussions of biological plausibility, as it addresses key issues from a perspective independent of specific particle composition. When different ventilatory regions of the lung are compromised in pre-existing conditions such as asthma, COPD or congestive heart failure with respect to their ventilatory capacity, those regions of the lung that are still healthy can receive a disproportionately high dose of particles. This, in turn, places those remaining healthy regions at even greater risk by further compromising the lungs' reserve capacity. An increase in ambient particle concentrations of 20-40 $\mu g/m^3$ would translate into relatively small increases in peripheral lung particle deposition. From a pathophysiological perspective, it seems unlikely that such particle concentrations could worsen ventilation-perfusion ratios in a healthy or injured lung to the point of producing hypoxemia sufficient to cause pulmonary edema due to left ventricular failure, increased permeability or malignant arrhythmia. Similar clinical arguments exist when attempting to relate exacerbations of respiratory infections to increased particle loads.

Particle exposure could increase susceptibility to infection with bacteria or respiratory viruses, leading to an increased incidence of death from pneumonia. Although pneumonia rarely results in death within 24 hours of onset, serious infections of the lower respiratory tract can develop and evolve over days or weeks. This would not support the observed acute, untimely mortality. It would, however, support the observed effects on increased respiratory infections seen in panel studies in children and adults. Moreover, hospital admissions should increase, and have been observed to do so, and the widespread availability of ventilatory support would tend to further blunt any immediate effect of particle exposure on pneumonia mortality. As yet no precise mechanism of action has been established which could explain individual deaths at the ambient particle levels measured in most of the recent epidemiology studies.

The involvement of ultrafine particles may provide a means to explain the epidemiological observations of rapid effects of low ambient particle concentrations in cardiorespiratory disease (Seaton et al., 1995; Oberdorster et al., 1995). The urban particulate cloud, part of which is derived from combustion sources, may contain up to millions of nanometre sized particles per ml or cc, with a gravimetric concentration of only 100-200 $\mu g/m^3$. The particle surface area is therefore greatly enlarged, and can carry adsorbed metals, acids, and toxic organic molecules down to the deep recesses of the lung. Ultrafine particles are not as readily phagocytized and are therefore, more likely to react with the lung and be transferred to the interstitium. Seaton (1995) therefore proposed that ultrafine particles could be responsible for the adverse effects seen in the epidemiology studies, due to their actions in provoking alveolar inflammation, which leads to changes in blood factors and coagulability, thus causing the release of mediators able to provoke attacks of acute respiratory illness in predisposed individuals.

These suggested biological mechanisms, while still requiring more research and confirmation, help to close a major gap in our understanding, thus providing some support for the idea of causality. Precise mechanisms of action have yet to be established, although they are only one component in establishing a causal relationship between particulate matter and mortality and morbidity impacts.

***Coherence** (Is the effect seen in a variety of related endpoints as could be expected?)*

The large series of epidemiology studies have demonstrated a "pyramid of effects" for outcome severity, frequency and extent of public health impact associated with particulate matter. Many studies have associated PM with cardiorespiratory mortality. Increased mortality is generally associated with increased hospitalizations and one would expect that hospital admissions would also be elevated, to a greater degree than mortality, since not all affected people would die. Similarly, emergency department and doctors' visits, respiratory symptoms, lung function, respiratory-related reduced activity, and days' absent from work or school due to respiratory illnesses would be expected to be elevated. All of these have in fact been observed. In addition, the slopes of the response curves also tended to increase as the effects went down the scale of seriousness on the "pyramid of effects", which adds to the coherence.

Therefore, a robust pattern of coherence between endpoints both qualitatively and quantitatively is provided by the associations shown between particulate matter and a broad range of endpoints from the least to the most serious.

***Alternative explanations and the possibility of confounding** (Should the observed associations be considered to be causal or to reflect the effects of uncontrolled confounding or other methodological artifacts?)*

Confounding factors of potential concern in the analyses of health impacts and daily fluctuations in particulate matter include seasonal – yearly – and/or day-today trends, temperature, and epidemics of infectious diseases, especially influenza. Weather, which can be highly correlated with particulate matter, exerts an independent, often stronger effect than PM on health endpoints such as mortality or hospitalizations. Most of the mortality and hospitalization studies controlled for temperature in the analysis in a variety of ways, and most also took the non-linear relationship of temperature with disease into account. Other meteorological variables, most frequently humidity or dew point temperature (which is related to humidity), were usually also incorporated. Some studies (e.g., Schwartz 1993, 1994, 1996; Ito and Thurston 1996) undertook sensitivity analyses to ensure that weather was not unduly influencing the results. Associations were observed in warmer and colder climates, dryer and more humid or maritime climates, and in locations where peaks occurred in the winter (Europe) as well as others (US and Canada) where summer peaks due to vehicles and secondary photochemical action were observed. The method of control did not seem to influence the results very much and the consistency suggests that climatic factors have been adequately taken into account and are not important confounders of the PM-disease association.

Epidemics of infectious diseases, particularly influenza, can bias the particle-adverse health relationship if unrecognized. Epidemics of influenza or another infectious disease could explain the association of particulate pollution with mortality only if the higher concentrations of particles invariably corresponded to times with outbreaks. In some analyses, modelling was also used to control for confounding by known influenza epidemics or by calendar year as a surrogate. It also seems unlikely that some weather-related or methodological confounding factor, known or not yet identified could be responsible for the positive associations of PM and mortality and morbidity in such a wide variety of locales.

Perhaps the most difficult question to deal with regarding the possibility of confounding has been the role played by other gaseous pollutants (measured or unmeasured) and the concern that the association which has been ascribed to particles is actually reflecting the association of one or more of these other air pollutants. Airborne pollution always occurs as a mixture of agents, of which particulate matter is only one. The gaseous pollutants ozone, SO_2, NO_2 and sometimes CO are those usually considered. Since the sources of the gaseous pollutants are often the same as those for particulate matter, their correlation coefficients can be very high ($r \geq 0.8$) in some locations. When strong correlations between the pollutants are present, disentangling their effects in regression analyses is very difficult or even impossible. In such cases, some authors (Dockery et al., 1996; Schwartz et al., 1996b) recommend that the best course of action is to rely on comparison of the PM effect in many different locations where the pollutant mixtures are all different.

Since other pollutants may also exert effects on the health outcomes, they may bias the results, and/or the regression coefficient for PM can include the effects of the confounder (these can be negative as well as positive). If other covariate pollutants are not examined at all in the analysis, the suspicion exists that any effect observed is due to the unexamined covariate instead of the pollutant of interest.

The agent most often associated with mortality and hospitalizations independently of particulate matter is ozone. Some studies did not consider ozone in their analysis, leaving open the possibility that part of the effects ascribed to particles could have been due to ozone, particularly for studies looking at summertime effects, when ozone can be high. In addition, some investigators have used 24 hour ozone measurements which do not reflect the nature of the response to high diurnal ozone peaks, and tend to hide the "signal" of this strongly cyclic pollutant from the background noise. In North America, SO_2 is not usually highly correlated with PM, and it generally has not been found to be a predictor of mortality or increased hospitalizations in North American studies. NO_2 and CO were only occasionally examined or regressed together with PM. The almost invariably high NO_2-PM correlation coefficient implies some instability of results when both are considered together. CO appeared to have some association with mortality and hospitalizations that was independent of any effects due to PM (Burnett et al., 1995; Pantazopoulou et al., 1995).

In analyses designed to help separate out the effects of one pollutant from another, the association of particulate matter with adverse health outcomes reported in the epidemiology literature was remarkably robust to inclusion (one at a time) of all four of the normally present gaseous air pollutants (SO_2, NO_2, CO and ozone). Moreover, the magnitude of this association was often (but not always) greater than any of these other air pollutants individually or combined. The magnitude, robustness, and consistency of this association across so many locations with differing air pollutant mixtures supports the position that particulate matter of some kind is the best indicator for the effects of air pollution on adverse health outcomes.

Particulate matter is the only regulated air pollutant whose chemical nature is unknown. Moreover, in different locations, and even in the same location at different times, its physical size and chemical properties differ, depending on its sources, which can be both local and regional, via long-range transport. In addition, the various metrics that have been and continue to be employed for measuring particulate matter make comparisons between studies more difficult, if not impossible, in some cases. Particulate matter with a median aerodynamic diameter (d_a) less than 10 μm (PM_{10}) and matter with a d_a of ≤ 2.5 μm have been emphasized in this review since they represent the size fractions with maximum penetration to the thoracic and pulmonary regions of the respiratory tract respectively, and thus are the biologically relevant fractions. Review of the finer fraction of PM_{10} indicates that one or more of these are probably more closely linked than PM_{10} to adverse health outcomes (Schwartz et al. 1996; Vedal 1996). $PM_{2.5}$, a subset of PM_{10}, is at this time the best general indicator of the pulmonary toxicity of the fine fraction of PM_{10}.

In the past few years, many studies using this metric in addition to PM_{10} have become available. Other fine fraction metrics include sulfates and particle strong acidity (H^+), two more chemically defined inorganic species, also measured as mass. Although these two measurements have provided strong associations with adverse health outcomes in certain locations, and have been suggested as the metric of choice to act as a surrogate for fine particles in standard-setting (Lippman and Thurston 1996), they are too area-specific to be used as general metrics for regulatory purposes (i.e., not all areas experience elevated levels of sulphates or aerosol acidity). Other optical-based fine particle measurements include Black Smoke or British Smoke (BS) (widely used in Europe), coefficient of Haze (CoH), used in Canada and formerly in some areas of the US, and km, a visibility measurement used in California. Although these are difficult or impossible to correlate with mass measurements, they have been qualitatively useful in correlating particle exposures with adverse population health effects, and in providing some further insights into the toxicity of fine particles. Although considerable work is being done with ultrafine particles on the biological mechanism of action in the etiology of disease, only one or two epidemiology studies have attempted to (or been able to) examine ultrafine particles either as mass or as a numerical value.

In a number of recent studies, several mass-related particle measurements including two or more of TSP, PM_{10}, $PM_{2.5}$, $PM_{10-2.5}$ (PM_{10} coarse fraction), sulphates, or particle strong acidity (H^+) have been investigated together trying to pinpoint more precisely which component is responsible for the adverse effects observed. These metrics all tend to be highly correlated with each other (correlation coefficients are usually over 0.6, and range as high as 0.99), partly because they arise from the same combustion sources. Even the best and most statistically powerful multiple regression analysis cannot separate out the effects of two or more closely correlated agents with any degree of certainty, and they cannot successfully be co-regressed. However, the database of locations is now sufficiently large, with differing pollutant mixes, that we are beginning to be able to detect which of these metrics are more important from the health point of view. Tables 12.7-12.9 present results from some studies in which fine and coarse particles were regressed together. Thus far, we can conclude that fine particle metrics generally appear to be more predictive of health outcomes than coarse particle metrics, although in some locations, coarse particles remain important and cannot at this time be dismissed.

13.4 CONCLUSIONS – CAUSALITY REVISITED

The evaluation of the causal nature of a relationship without supporting results from direct experiments is neither easy nor entirely objective. While it is generally accepted that statistical associations drawn from well-conducted, randomized experimental studies provide the strongest evidence for causal relationship, little evidence is available from the non-epidemiology studies. However, epidemiological observations have often preceded the biologic knowledge of the day. Examples include the first epidemiological studies linking smoking and lung cancer, the transmission of cholera by drinking water, and the efficacy of cowpox vaccine against smallpox, the latter predating knowledge of virology by almost 200 years. A fundamental purpose of epidemiology is to establish a cause for observed adverse effects on public health with enough certainty that taking appropriate action to mitigate those effects will be possible.

According to the evidence presented in the preceding sections the strength and consistency of the epidemiological evidence for mortality and morbidity effects at current levels of particulate air pollution is remarkable, robust, consistent and compelling. Particulate matter, as both PM_{10} and $PM_{2.5}$, has been associated, in a large number of well run daily or short-term time-series studies and long-term cohort studies, with mortality involving both respiratory and cardiac diseases, cardiorespiratory hospitalizations, increases in respiratory symptoms such as chronic bronchitis and decreases in lung function and lung capacity in children after both acute and longer term exposure, reduced activity due to respiratory illnesses, and work or school absences. A strong pattern of coherence is provided both qualitatively and quantitatively by the associations shown between particulate matter and a broad range of endpoints from the least serious to the most. Although the magnitude of the estimates of increased risk are seemingly small, they were often highly statistically significant. Moreover, the adverse health effects represent a large impact on the general population, since most of the population is exposed. The time pattern of exposure and effect adds to the coherence of the picture, with the exception of the rapidity of the effects on mortality (preliminary mechanistic research may soon provide an explanation for this observation). It should be noted that biologic plausibility is not an absolute requirement for a conclusion of causality.

The evidence is considered to be strong regarding the specificity of the effect for respiratory and cardiovascular outcomes. While alternative explanations for these associations could be the presence of unmeasured confounders and/or highly correlated covariates, a number of studies have reduced, but have not eliminated these concerns altogether. On the other hand, these associations have been noted across a wide range of exposure levels and pollutant mixtures in a wide variety of locations. This gives some confidence that confounding cannot be responsible for all of the associations which have been observed. The evidence is hard to judge, but on balance, is sufficient enough to conclude that particulate matter per se is associated with adverse health effects.

The data provide little evidence of a threshold, with estimates of mortality and morbidity increasing with increasing particle concentrations. That is, any increase in ambient particulate matter is associated with a statistical increase in adverse health effects. On a population basis, the hypothesis is that what we're seeing is exacerbation of pre-existing disease, or enhanced response of a subpopulation of sensitive individuals. Pre-existing disease includes COPD (chronic bronchitis, emphysema, asthma, pneumonitis), upper and lower respiratory tract infections, influenza and health disease.

While caution is necessary in judging relationships to be causal, this should not be used simply as a cover for dislike of the consequences of accepting such a relationship. Although the mode of particulate action is not clear, the coherency, consistency and temporal pattern of the evidence of different health outcomes strongly suggest a causal relationship between PM pollution and cardiorespiratory outcomes. There remains no good understanding on what is the most "biologically" relevant index of particulate matter, although there is sufficient evidence to conclude that the causal relationship between particulate matter and cardiorespiratory disease is drive by the fine particle fraction ≤ 2.5 μm.

13.5 RECOMMENDATIONS FOR RESEARCH

- Further research, both *in vitro* and *in vivo*, is needed on the basic mechanism(s) of toxicity of particles to elucidate a biologically plausible mechanism of action of particles. This need is critical in view of the provisional nature of conclusions on causality from the indirect evidence provided by the ecological epidemiology data set.
- Animal studies are required that examine the interactions between particles and the gaseous pollutants, in particular ozone, which has a well-defined mode of action shown to have associations with adverse respiratory health independent of those due to particulate matter.
- Further epidemiology studies are needed to investigate the association between exposure to particles and acute effects on health to determine which component (size, number or mass) of the particle mixture has the greatest effect on health.
- The effects of all the combustion-related gaseous pollutants on the associations between particulate matter and mortality and hospitalization requires further investigation, in view of some preliminary indications that these are critically important.
- Studies of the effects of hourly variations in particles levels on health outcomes should also be undertaken.
- Research is needed on the composition and physical-chemical characteristics as well as on the behavior of the ultrafine particles occurring in the urban environment under different meteorological conditions.
- Additional PM_{10} data is required to allow characterization of the extreme (upper portion) of the frequency distribution and determine what processes are responsible for events leading to extreme concentrations of particles.
- Continued expansion of the PM_{10} monitoring network, particularly size selective sampling, in Canada is recommended.
- Collection of data on shorter time frames is recommended to enhance our understanding of exposures.
- Additional information is required on the relationship between particle levels and indicators of morbidity (symptoms and pulmonary function changes) to fully establish coherence across the entire range of demonstrated and expected health outcomes associated with exposure to PM_{10}.
- Research is also needed to better understand the limited data that suggests personal exposures often exceed estimates of exposures from fixed samples by a significant factor.
- More research is needed on the chronic effects of long-term exposure to air pollution.
- Research on the cardiopulmonary effects of particle number and composition, rather than simply particle mass, is also needed.

14 IDENTIFICATION OF THE REFERENCE LEVELS FOR PARTICULATE MATTER

The Federal/Provincial Working Group on Ambient Air Quality Objectives and Guidelines is required to identify an ambient Reference Level, defined as a level above which there are demonstrated effects on human health and the environment. It provides a basis for establishing goals for long term air quality management. This Chapter addresses the scientific information that forms the basis for the development of this level in this Chapter.

No dose-response relationship has been identified for the effects of particles on vegetation, nor have any no effect- or lowest-observed adverse effect levels (NOAELs or LOAELs) been identified. Much of the evidence is related to particle composition rather than the effect of particle size. Therefore, the Working Group will not identify a Reference Level for vegetation. Likewise, the paucity of data related to particulate matter and effects on materials precludes the identification of a Reference Level for material impacts.

The effects of particulate matter on visibility are due primarily to the fine particle fraction. Therefore, defining a Reference Level to protect visibility is not appropriate based on PM_{10}. Defining an acceptable level of visibility, or an index of change in visibility that is reliably and uniformly perceived by the public, however, is extremely difficult. Therefore, the Working Group cannot identify a $PM_{2.5}$ Reference Level to protect visibility at this time.

Subjective measures of respiratory health such as school absenteeism, days of work loss and restricted activity are usually collected through survey instruments. The data on these measures are valid indicators of respiratory health, however, because the data is limited, they are not appropriate indicators on which to base the identification of a human health Reference Level. Spirometry measures small and reversible changes in lung function, a robust measure generally. Again, the limited experimental data also prevents the identification of a Reference Level.

The relationship between both PM_{10} and $PM_{2.5}$ and cardiorespiratory hospital admissions and mortality examined via large administrative databases is the strongest both qualitatively and quantitatively.

According to the weight of evidence presented in Chapter 13, the strength and consistency of the epidemiological evidence for mortality and morbidity effects at current levels of ambient PM are remarkable, robust, consistent and compelling. The associations between PM and both mortality and hospital admissions are consistent across geographic locations, including many cities in North America, Europe and South America, where sources and concentrations of PM are different. As each individual who dies or is admitted to a hospital must have a diagnosed and recorded condition, the data could be considered more reliable for deriving a Reference Level than subjective measures of particle impact. Another key feature of the hospital admissions studies is that they allow for the measurement of effects in all age groups. A significant number of hospital admissions are seen across all age groups. A greater number of mortality studies, both acute and chronic, are available, and several of these are also statistically robust and are based on large administrative databases. However, it is very likely that the daily hospital admissions studies capture a larger segment of the affected population than do the daily mortality studies.

Positive associations have been observed between particulate matter and both daily mortality and hospitalization rates with no clear evidence of a threshold level. That is, any increase in ambient particulate matter is associated with a statistical increase in mortality and hospitalization rates. The lack of evidence of a threshold precludes the possibility that a sufficiently low level of exposure will be free of any degree of impact. Therefore, a Reference Level identified from this data will in fact be an estimate of the lowest ambient PM level at which statistically significant increases in health responses can be detected and not a level where impacts will not occur.

Dose-response information derived separately from the linear regression slopes was available from quintile/quartile analyses, or from nonparametric regressions, from five US and one South American daily mortality studies and three daily hospitalization or emergency department visit studies. None of the studies assumed any particular shape for the dose-response curve. Most of the studies presented information on PM_{10}.

Quintile analysis of three daily mortality studies from the US (Dockery et al., 1992; Pope et al., 1992; and Schwartz, 1993) showed an average PM_{10} 24 h LOAEL of 35 $\mu g/m^3$ (relative to quintile one, which is recognized as not necessarily a level without adverse effects). After weighting (by one standard error) to give more weight to studies with tighter confidence intervals, the estimate decreased to 30 $\mu g/m^3$ averaged over 24 h. Since the quintile values depend on the average concentration for each location, eliminating cities with higher average concentrations from consideration in establishing a LOAEL is reasonable. Therefore, the Brazilian city, Sao Paulo was not used further to help define the Reference Level. The data from Lyon et al. (1995) were also not used because of discrepancies in its quintile analysis and a faulty overall analysis.

The US six-cities daily mortality study provided additional information on each of the six cities individually, and a combined analysis. The latter analysis revealed a combined 24 h mean PM_{10} LOAEL of 25 $\mu g/m^3$ (Schwartz et al., 1996). In Boston MA, the estimated 24 h LOAEL was also 25 $\mu g/m^3$ compared with Portage, WI, where no association was found between mortality and PM_{10}, and the estimated 24-hour mean ambient PM concentration was 18 $\mu g/m^3$. The lowest level at which statistically significant increases in mortality were detected in the Schwartz study (1993) was 20 $\mu g/m^3$ PM_{10} averaged over 24 h based on a nonparametric dose-response curve with monotonic increases in mortality associated with PM_{10} concentrations ranging from 10 to 140 $\mu g/m^3$. Thus, three possible Reference Levels, 20, 25, or 30 $\mu g/m^3$ averaged over 24 h, are suggested by the mortality data. Of these, the mid value of 25 $\mu g/m^3$ is given considerable weight due to the high quality and the size (duration times responses) of the study on which it is based.

Similar types of analysis were done for hospitalizations or emergency department visits, providing LOAELs, at the second quartile of ambient PM_{10} data, of 20 $\mu g/m^3$ (Schwartz et al., 1993), 36 $\mu g/m^3$ (Schwartz, 1994b; Schwartz and Morris, 1995) and 4.13 $\mu g/m^3$ SO_4^{2-} (equivalent to 25 $\mu g/m^3$ PM_{10} based on site specific conversion factors, Burnett et al., 1995). Nonparametric dose-response plots for increased pneumonia and chronic obstructive pulmonary disease (COPD) admissions (Schwartz 1994a), and a decile plot for increased admissions for respiratory disease (Burnett et al., 1994) provide support for a 24 h LOAEL of 20-25 $\mu g/m^3$. Of the hospitalization studies, when identifying the Reference Level, considerable weight is given to the southern Ontario study, which identified a LOAEL for PM_{10} of 25 $\mu g/m^3$ (Burnett et al., 1995). This study is one of the largest of its kind (which lends statistical power to the study), the analysis is robust, it used six years of daily sulphate measurements (an excellent surrogate for fine particles and a reasonable one for PM_{10}), and encompassed a large geographic area in Canada.

Thus, both the mortality studies and the hospitalization studies support the identification of 25 $\mu g/m^3$, averaged over 24 h, as the Reference Level for PM_{10}.

Deriving Reference Levels for fine particles is more difficult because of the paucity of studies which examined fine particles, especially by nonparametric methods or quartile/quintile analysis. While many European studies examined British Smoke Shade or Black Smoke (BS), which measures fine particles with mean aerodynamic diameters of approximately 5 $\mu g/m^3$, none could provide site-specific conversion factors for $PM_{2.5}$. The choice of $PM_{2.5}$ as a measure on which to base the fine particle Reference Level was originally made to account for the bimodal nature of particles in the atmosphere, and for their bimodal deposition in the body. The fine fraction, with a cutpoint of 2-3 µm aerodynamic diameter, is preferentially deposited deep in the lungs compared with the larger particles that are deposited higher up in the respiratory tract. It is unlikely that conclusive epidemiological evidence will be forthcoming in the near future that would argue for abandoning $PM_{2.5}$ monitoring in favour of an even smaller size fraction. Non-mass measurements such as number counts or optical-based measurements have been suggested as appropriate metrics on which to base Reference Levels, however, they have rarely been used in epidemiology.

$PM_{2.5}$ has been most clearly associated with adverse health effects in the epidemiological studies, and overall, in most but not all studies, $PM_{2.5}$ has been shown to have a robust association with mortality. In addition $PM_{2.5}$ can be considered a general surrogate for fine particle effects in all regions of the country, where fine particle concentration and composition vary widely. While particle strong acidity (H^+) is an attractive choice of metric because of its biological plausibility as a toxic agent, it has proven to have an inconsistent association with adverse health endpoints.

The six-city mortality study, given considerable weight in the PM_{10} analysis, also provided LOAEL estimates for $PM_{2.5}$ (Schwartz et al., 1996). Over the entire six cities, the 24 h median LOAEL was 14.7 $\mu g/m^3$ for $PM_{2.5}$, with the association between daily mortality and $PM_{2.5}$ being highly significant. The slope of the concentration-response curve was as steep or steeper at lower concentrations than at higher ones. Three of six cities individually showed associations between mortality and $PM_{2.5}$ (24 h mean LOAELs were 15.7 $\mu g/m^3$, 18.7 $\mu g/m^3$ and 20.8 $\mu g/m^3$). Two cities with low mean 24 h $PM_{2.5}$ concentrations (11.2 $\mu g/m^3$ and 12.2 $\mu g/m^3$) showed no significant associations between daily mortality and $PM_{2.5}$, while the association in the sixth city was only marginally significant due to confounding by coarse particles, for which the correlation coefficient with $PM_{2.5}$ was high (r=0.69). The latter three cities were not considered further. Thus the composite LOAEL from this study, and the three individual city results, with significant associations, reveal LOAELs ranging from 14.7 $\mu g/m^3$ to 20.8 $\mu g/m^3$, averaged over 24 hours.

The only hospitalization study that provided a quartile or decile analysis using any fine particle measure was the Burnett et al. (1995) study on cardiorespiratory admissions in southern Ontario. In an analysis similar to a quartile analysis, the lowest concentration at which a statistically significant increase in cardiorespiratory hospitalizations was observed was a mean sulphate value of 4.13 $\mu g/m^3$. This is equivalent to a mean $PM_{2.5}$ concentration of 15 $\mu g/m^3$, based on site-specific conversion factors. A decile analysis was also provided, showing a curvilinear relationship down to zero, with a near-linear steep slope at all levels below 6 $\mu g/m^3$ SO_4^{2-}. As noted in the rationale for PM_{10} Reference Levels, this was a well designed and carried out study with sufficient power to give confidence in its results. Since SO_4^{2-} and $PM_{2.5}$ are usually highly correlated, in the range 0.7-0.9 in southern Ontario, the SO_4^{2-} measurement is considered an excellent surrogate for fine particles in this study.

Two recently published chronic cross-sectional studies showing decrements in lung function (Raizenne et al., 1996) and increases in respiratory symptoms (Dockery et al., 1996) also provide a limited measure of support for adverse effects at the mean 24 h LOAEL of 14.5 $\mu g/m^3$ (range 5.8 to 20.7 $\mu g/m^3$).

Thus, the mortality and morbidity (hospitalization, lung function, and respiratory symptoms) data point to 24 hour average of 15 $\mu g/m^3$ as a $PM_{2.5}$ Reference Level.

While mortality and hospitalizations have been emphasized because of their ability to provide some measure of quantification, they are only the tip of the iceberg with respect to other adverse health effects including exacerbation of respiratory symptoms, reduced lung function, restricted activity due to illness, loss of workdays or school absenteeism, and increased medication use. These particulate matter-adverse health associations, which the Working Group believes to be causal, are observed at the concentrations currently occurring in Canada.

Recognizing the coherent, consistent evidence for a continuum of effects associated with PM, the Working Group recommends that the Reference Level for particulate matter less than, or equal to 10 µm (PM_{10}) be 25 $\mu g/m^3$, averaged over a 24 h period, and that the Reference Level for particulate matter less than or equal to 2.5 µm ($PM_{2.5}$) be 15 $\mu g/m^3$, averaged over a 24 h period. Both Reference Levels are estimates of the lowest ambient PM level at which statistically significant increases in health responses can be detected based upon available data and current technology. The identified Reference Levels should not be interpreted as thresholds of effects, or levels at which impacts do not occur.

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